CLUES IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE

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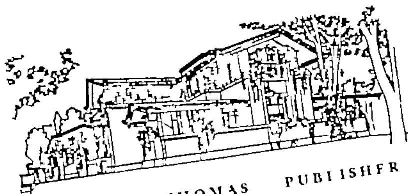
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PREFACE

A LONG experience in the practice of medicine with particular attention to cardiovascular disease has revealed the value of a search for diagnostic, prognostic and therapeutic clues often not listed as such in the current textbooks or general articles. Although such clues have frequently been discussed and analyzed in individual researches and published papers they have not been assembled for ready reference During the last 10 years or more I have been in the habit of giving a lecture on the subject in graduate courses in heart disease The usefulness of such a presentation has resulted in a request that I expand it for publication into a small volume

The chapters in this book present clues obtained from general observation of the patient from his past and family histories from his symptoms from signs obtained by physical examination auscultation and sphygmomanometry and the results of x ray and electrocardiographic studies. Since this volume is intended largely for the general practitioner I have included very little of the findings obtained by the more specialized laboratory procedures such as cardiac catheterization angiocardiography ballistocardiography phonocardiography and biochemical technics. For a discussion of the findings in such technics the reader is referred to the larger or special volumes concerned therewith

Before completing the manuscript I have collected from various medical colleagues in this country and abroad word about some of their own pet clues and thus, through their kindness I have reinforced my own experience with theirs. For this I am indebted to the following physicians as

well as to many others, too numerous to name here whose writings I have read or whose lectures I have attended

Drs H Alessandri Santiago Chile R. Aixala, Havana, Cuba E. Cowles Andrus Baltimore Md R. Armas-Cruz, *Santiago Chile* *Arlie Barnes, Rochester Minn D Evan* Bedford London England Julien Benjamin Cincinnati Ohio Edward F Bland Boston, Mass. Geoffrey Bourne London, England George Burch New Orleans, La. C. Sidney Burwell Boston Mass. Maurice Campbell London England J H Cannon Charleston S C. Pedro Castillo Havana Cuba Francis Chamberlain San Francisco Calif Ignacio Chavez, Mexico City L. Condorelli Rome Italy Pedro Cossio Buenos Aires Argentina Clarence de la Chapelle New York, N Y Lewis Dexter Boston Mass. Eugene Drake Portland Me Pierre Duchosal Geneva Switzerland Thomas Durant Philadelphia Pa Laurence Ellis, Boston Mass. William Evans, London England Harold Feil Cleveland Ohio Marshall Fulton Providence *R I L. Gallavardin Lyon France George* Griffith Pasadena, Calif Burton Hamilton Boston, Mass. Tinsley Harrison Birmingham Ala. John Hepburn Toronto Ontario George Herrmann Galveston Texas T Duckett Jones New York N Y William J Kerr San Francisco Calif Robert King Seattle Wash Charles Laubry Paris, France R. F Leinbach Charlotte N C. J Lenegre Paris France Samuel Levine, Boston Mass. Robert L. Levy New York N Y Camille Lian Paris, France David Littmann Boston Mass. R. Bruce Logue Emory Ga Genival Londres Rio de Janeiro Brazil Kempson Maddox, Sydney Australia E. Magalhaes-Gomes, Rio de Janeiro Brazil I Mahaim Lausanne Switzerland Benedict Massell Boston Mass. Edwin P Maynard Brooklyn N Y Roberto Menezes de Oliveira Rio de Janeiro Brazil Johnson McGuire Cincinnati Ohio Hugh Morgan Nashville Tenn Gustav Nylin, Stock

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Some of the more epigrammatic observations contained in the replies of these medical friends I am including as direct quotations generally verbatim I appreciate very much the permission to do so.

Also I would like to express my appreciation to the editors of these volumes Drs Irvine Page and A. C. Corcoran for asking me to write this book in the first place to my secretary Miss Helen Donovan for preparing the manuscript, to Miss Louise Wheeler and Dr Allan Friedlich for assembling the illustrations and to the publishers for their friendly coöperation

PAUL D WHITE

Boston Massachusetts

CONTENTS

LIST OF ILLUSTRATIONS

Ch pter II

THE X-RA SHADOW OF THE H RT AND GRE VESSELS

Chapter 12

Electrocardiographic Cuts

Figure

CLUES IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE

INTRODUCTION

THE PRACTICE of medicine has several attractive features which render it one of the most fascinating adventures in the world. In the first place it deals with people an absorbing occupation in itself both because of its humanitarian role and because of the infinite variety of personalities involved. One must individualize strictly and study and treat the whole man as well as the disease (in this case of his heart) In the second place it is a scientific discipline which has greatly advanced in factual knowledge during the last few decades and allows us now to bring hope to many patients who were doomed a generation ago And, finally it is very often a fine art of detection which requires for its solution an assembly of revealing clues The well rounded practicing physician needs therefore in his own person a fair share of all these three attributes 1) the love of humanity 2) a scientific mind and 3) an interest in the unraveling of mysteries. A busy doctor hardly needs to spend much time reading detective stories for his entertainment for he is faced by many problems of this sort in his daily life

The diagnosis and treatment of heart disease lend themselves especially well to the gathering of clues for elucidation. Even what seem like straight forward lesions may in their evolution develop unexpected complications which might have been foreseen had certain clues been looked for and noted along the way And not infrequently there are obscurities at the very onset that can be cleared by a conscious search for clues. In the past, although systematic questioning and examination of patients may have been

the customary rule there has been on occasion little effort to separate the red herrings from the important clues. There has tended to be a haphazard recording of facts without emphasis on certain significant clues which can be scientifically appraised. A further point of considerable importance is that one cannot rely on the absence of symptoms or signs unless they have been specifically inquired into or looked for. A doctor who cannot take a good history and a patient who cannot give one are in danger of giving and receiving bad treatment. And finally, In diagnosis one should seldom think of any *single case* one may have seen. This often warps the judgment as Robert Hutchison has mentioned, and yet it is true that very rare causes of disease such as amyloidosis and neoplasm should be thought of in very obscure cases. Do not, however yield to the natural temptation to make a snap diagnosis.

CHAPTER I

GENERAL CLUES

THE PHYSICIAN who himself sees, questions and examines a patient has a great advantage over the one who simply reads or hears the patient's record prepared by someone else. Not, of course, that he could have necessarily prepared a better record, but there are some things in dealing directly with a person that are hard to put down on paper. It is this disadvantage that prevents a complete picture of a case in a clinicopathological conference, although that difficulty is counterbalanced by the fact that the detective on the case can usually count on the probability of a rare or unusual diagnosis.

The general appearance of the patient and his or her mental attitude are of the greatest importance at the very outset in the appraisal of a case and though quickly noted by the observer are rarely recorded. They are clues for the correct interpretation of symptoms when they are described by the patient and have a bearing on prognosis as well as on diagnosis and treatment. Not only do they help eventually in outlining treatment but they suggest quite early whether or not it will be easy or difficult to secure the confidence of the patient and therewith a full coöperation in carrying out treatment which often must extend for a good many years.

The physician must himself of course sympathetically receive the patient and listen to the story before trying to impose his own personality The very first hour of consultation may make or mar all future relationships and render easy or difficult the establishment of a correct diagnosis and proper treatment In accomplishing this there is a happy mean between a studied cold objective manner and a maudlin over-solicitude though each case must perforce be judged and treated on individual merits.

Even the doctor s secretary who makes the first appointment by telephone or direct conversation or a friend or a member of the family or another physician who refers the patient, may not infrequently be able to add comments of considerable importance about the physical or mental attitude of the patient before the doctor himself meets him these should not be ignored though they may need to be corrected later on better acquaintance

Is the patient impetuous or overanxious or is he slow in seeking advice and tending to minimize his troubles? The first few minutes of the consultation afford such information which is of course essential in the elucidation as well as in the appraisal of the symptoms. Is he nervously sensitive with a low threshold and so more conscious of his trouble whether pathological or physiological or a mixture of both? Or is he callous about himself or others and so insensitive that some disease process may be fairly advanced before he is actually aware of it or willing to acknowledge that he is sick? It is sometimes difficult to define these attributes of the patient but even more is it true that they are infrequently put down on the case record although they are appreciated and actually subconsciously taken into consideration by the doctor himself

Moreover what is the extent of the patient s knowledge and insight into his medical status? How much has he been

told by other doctors by members of his family and by friends? And how much of what he has been told is true or does he believe? Is he a reader of medical columns in newspapers or of medical articles for the layman in popular magazines? Does he read medical books in the libraries or at home? How rich is his vocabulary in medical terms? How much does he actually know of these terms and how much does he apply them in his own case and how justifiably? Does he admit that he thinks that he missed his calling and should have been a doctor? Or did he have a little medical education himself at some time or have there been doctors in his family? Before completing a history of the case it may be worth while for the doctor to ask a question or two along these lines which may be very revealing On more than one occasion I have encountered lay patients who were reading my own textbook, sometimes however the wrong chapters therein.

It also happens sometimes that the most difficult patient of all is the physician himself especially an internist, who imagines all the worst possibilities at the very beginning. On the other hand some of my most coöperative patients have been doctors, even cardiologists, who have delivered themselves for diagnosis and treatment without prejudice or doubt.

Before closing this chapter on these clues in general the family and friends of the patient must be taken into consideration. What is their attitude about him or his illness. It is well to obtain word of it directly from them as well as from the patient. Such information can be gleaned by letter by telephone or best by personal conversation which need not arouse the patient's suspicion. In obtaining this information the family and friends must be judged just as is the patient as to emotional attitude veracity and details of the situation at home, at school or at work. Of

course much of this can be obtained best of all from the good old fashioned family doctor if there is one As in the case of the patient himself are the family associates in school or at work and close friends over-solicitous or are they impatient and even inconsiderate? A summary of all this might well be incorporated confidentially in the patient's record.

Conscious *malingering is in my experience rare in* routine practice but, of course there are many instances of exaggeration of symptoms when disability insurance or payments are concerned and much careful evaluation of the overall picture is essential with as many objective data perhaps serially obtained as can readily be secured, since *injustice is possible both ways, by minimizing symptoms as* well as in their exaggeration We must always remember that a very neurotic person can be seriously indeed even dangerously ill with coronary insufficiency hypertensive cardiovascular disease or rheumatic valvular deformities and their effects One should be especially careful therefore to examine a patient more *thoroughly* than usual before excluding organic heart disease and diagnosing instead cardiac neurosis. This is said to be particularly true in the case of the full blooded Negro who may be seriously ill with only a little misery in his chest the significance of which may at first be underestimated

I would add that it is in general wise to take the patient into one's confidence if he is of average intelligence and can be trusted. For him to know what is wrong tends to rid him of unnecessary fears of both the unknown and the known to enlist his coöperation in his treatment and to uncover further clues in the future which may prove to be of inestimable help in his prognosis and further management. I have rarely if ever regretted such a step but of course much wisdom tact and experience are needed in

the proper presentation of his cardiac status to the individual patient himself

In addition to what has been said above there are several general clues that are more or less taken for granted in the appraisal of a patient but which deserve brief mention. The first is *age* A baby with something wrong with its heart is naturally suspected of having some congenital defect a child over four may have either rheumatic or congenital heart disease a young adult may have also thyrotoxicosis, early hypertension or even coronary heart disease in middle age all these kinds of heart disease are possible plus the syphilitic and the cor pulmonale while in old age congenital defects and rheumatic valvular disease are much less likely coronary heart disease and the effects of hypertension predominating senility itself does not carry with it any specific type of heart disease but often is beset by one of those just mentioned—rarely if ever does a person die of old age alone without adequate physical cause discovered at autopsy

Sex is an important clue in youth and middle age the more serious hypertensive cases and almost all patients with angina pectoris and coronary thrombosis are in those earlier decades males. The female hormone seems to be protective

Body build has at least one important correlation. It is primarily the mesomorph the wide stocky muscular male who is the typical early candidate for coronary heart disease And of course body build tends to be inherited, despite which fact we are searching for some protective measures in the way of life for such candidates.

The *state of nutrition* is often important. Obesity favors the so-called degenerative types of heart disease, that is, the hypertensive and the coronary while loss of weight and malnutrition suggest thyrotoxicosis, chronic crippling by

illness such as heart failure or even a prolonged effect of drug therapy as in the case of anorexia caused by digitalis.

The presence of *other congenital anomalies* always favors the diagnosis of congenital cardiovascular defects when there is doubt as to the cause of abnormalities of the heart or great vessels.

CHAPTER 2

THE PATIENT'S PAST HISTORY

THE PAST history of the patient is often full of clues and yet it is frequently the most neglected part of the entire examination. Haste and lack of systematic enquiry are usually responsible for such neglect. On occasion, however, it is due to a superiority complex, as in the case of a young hospital interne who may view his newly admitted patients as if they had never been examined before, ignoring the findings, opinion, and therapy of other doctors who have dealt with the patient before him as, for example, in some small hospital or in the practice of an L.M.D. or family doctor. Happily this custom is being steadily replaced by one of more respect for previous findings, expedited by the realization of the young doctors that they too will soon become the L.M.D.s of the future.

Sometimes the patient will himself supply important information about his past, but he is likely to forget certain items which are important so far as the heart and blood pressure are concerned, and which therefore should be asked about. For instance, have there been examinations for life insurance, when were they, and how carefully done? The amount of insurance applied for has a bearing since for small policies, as for five or ten thousand dollars, the examination may be very superficial while for large

amounts it may be very carefully carried out An interesting case of my own was a man 48 years old who came to see me in a panic because serious heart disease had just been discovered by an insurance examiner It turned out that he had a slight to moderate degree of patency of the ductus arteriosus, present of course since birth but diagnosed for the first time at 48 when he applied for a large insurance policy for business reasons. On this occasion he was carefully examined for the first time and the base of the heart was listened to as well as the apex, thus revealing the characteristic continuous murmur Previous insurance examinations for small sums had been quite inadequate the stethoscope was hastily applied to the cardiac apex only Incidentally this man is still alive and well carrying on a big business and playing golf without difficulty at the age of 77

Other physical examinations should also be enquired about as in school or industrial plant or for sports or military service Of course the results should be taken with a grain of salt as told by the patient but they may nevertheless be helpful and it may be possible sometimes to obtain the actual details of such examinations by consulting the records if they are available. On occasion it is of the utmost importance to see former electrocardiograms or x ray films for comparison with current ones. It may be difficult to obtain them but it is usually worth the effort and the diagnosis may actually be established by such comparison

The same thing is true about previous stays in other hospitals even in remote parts of the country or in foreign lands although of course the difficulties increase with distance and there may be considerable delays However a doctor s observations in some remote part of the world may hold the clue that clears some medical mystery and so should not be scorned.

To be more specific so far as the heart is concerned murmurs may date back to the first few weeks or months of life indicating the *probability* of a congenital defect or to the age of five or six following rheumatic fever scarlet fever or a severe sore throat Almost half of all cases of rheumatic heart disease present in adults give no history of having had acute rheumatism but this is not likely to continue to be the situation in the future in view of the increasing frequency of routine examinations of young children with the discovery of newly appearing heart murmurs by doctors in schools and in private practice especially on occasions when a child feels ill or appears to be so In very rare cases diphtheria may be the explanation for a v or bundle branch block rather than a congenital defect, coronary atherosclerosis or a myocarditis of unknown cause It is, however an interesting fact that almost all of my *own* young cases of coronary heart disease have had no serious infections of any sort.

One common *condition* that may be easily confirmed by clues in the past history is neurocirculatory asthenia (sometimes called anxiety neurosis) if such is suspected, leading questions are in order Has there been a story of fainting at adolescence or of a nervous heart, or of nervous prostration in early adult life? It is common for a victim of neurocirculatory asthenia to have had a checkered history of recurrent troubles of the sort

How much has the patient been told about his condition by other doctors and has he *interpreted* correctly what he has been told? Has he developed a fear of his heart disease or of symptoms which he thinks are due to heart disease but are actually caused by something else? Or has he become quite blase in his attitude or fatalistic with little faith in treatment or willingness to undergo it? Has there been a considerable difference of opinion about diagnosis

or treatment or both by various doctors whom he has con sulted while shopping around? And if there has been has he become depressed or disillusioned? Has he been driven to the new doctor who is yourself or to a new clinic by family or friends when he still has faith in his old doctor or in none at all? All these points should be settled at the very first opportunity if possible since they are of fundamental importance

Have there been operations of importance such as a cholecystectomy or prostatectomy or gastrectomy and how did the patient stand them? Was the heart considered normal at the time or was there special care in preparation or in anesthesia or in surgical technic? Were any of the operations designed to help the heart or other part of the circulatory apparatus, such as a pericardial resection for constrictive pericarditis commissurotomy for mitral stenosis shunt or other procedure for congenital defects or sym pathectomy for hypertension? How successful was the procedure and how long ago was it done? What were the de tails of blood gas and pressure analyses by cardiac catheterization before and after operation? If there has been a sym pathectomy is there still a relative postural hypotension which should be tested? How long did it last after the operation?

What about the diet? Has salt been restricted how much and for what (hypertension congestion)? How suc cessful was that salt restriction? How long was it carried out? Have there been any episodes of collapse or refractori ness to diuretic therapy due to sodium depletion. Have the serum electrolytes been well studied and serially? Have the calories been severely restricted, or but slightly in the therapy of obesity and how successfully? If the calories were much limited has there been a danger of vitamin lack or were vitamines given as an adequate supplement? Have

any bizarre diets been used and for what? And finally what has been the custom as to alcoholic beverages and tobacco? It is always wise to make specific mention of these items because several highballs a day may explain a failure to lose weight when food calories have been adequately re duced and arrhythmia (premature beats or paroxysmal tachycardia) can be the result of the effect of tobacco in a person whose heart is sensitive to it The amount of tea and coffee should also be ascertained for although no seri ous trouble comes from them they can be responsible for bothersome insomnia and palpitation.

Next what has been the actual drug treatment long ago and recent One should learn the details of this from pa tient or family previous doctors and hospital clinics. Once in a while there is merely a numbered prescription for some med cine without details as to contents this informa tion can be supplied by the pharmacist who put it up if the doctor who wrote it cannot be reached Some medicines alter the findings appreciably and so should be known For example digitalis in large dosage may depress con siderably the S-T segments of the electrocardiogram simu lating the effect of coronary insufficiency Quinidine can induce transient bundle branch block or change atrial fibrillation into flutter Certain drugs such as the veratrum alkaloids and hexamethonium can lower the bl od pres sure in hypertension. Thyroid and atropine or belladonna can raise the heart rate It is often important to know whether these medicine have been used in the past in what dosage and for how long and to ascertain whether they were helpful harmful or essentially inert Each one may be a clue t some present finding to some symptom or sign. And of course it is always important t know if there has been any narcotic habituati n

Finally so far as past history is concerned it is well to

know something about the past habits of work and play indulgment in strenuous sports heavy responsibilities in business family care or community adequacy of sleep relaxation and vacations and other aspects of the way of life that may have a direct bearing on current symptoms or signs. *Some recent heavy strain, physical or mental may* have precipitated acute trouble in a case of chronic heart trouble well tolerated till now Clues may be anywhere and need to be diligently sought. In the case of a woman the history should include questioning about the cata *menia, which may aggravate congestive symptoms and* signs and about the effect of childbirths. How many preg nancies have there been? Were they all well tolerated? Are all the children living? How old are they? And does the mother have adequate help at home?

CHAPTER 3

THE FAMILY HISTORY

OFTEN only lip service is paid to the obligation of every physician to know as much as possible about the family history of his patients. For the old time family doctor this knowledge was as a rule routinely acquired in detail during a long lifetime and was invaluable in his practice. In lieu of such a state of affairs it is important for the physician who takes care of cardiac patients or of any sick people as a matter of fact to record on his case histories accurate details so far as they can be secured of the ages whether still living or at the time of death of the near relatives of the patient being studied. These should include father, mother, brothers, sisters and children and on occasion grandparents too and half brothers and half sisters. Besides the information as to survival or death and the ages, important illnesses of these relatives should be noted, especially if such were the cause of invalidism or death or if they involved the heart or blood vessels.

Sometimes, particularly in the case of young people, family events are important to know about, for example unusual factors causing special strains like divorces, suicides, other violent deaths as in battle, accidents or concentration camps, bankruptcy, and the dependency of

know something about the past habits of work and play indulgment in strenuous sports heavy responsibilities in business, family care or community adequacy of sleep, relaxation and vacations, and other aspects of the way of life that may have a direct bearing on current symptoms or signs. Some recent heavy strain physical or mental may have precipitated acute trouble in a case of chronic heart trouble well tolerated till now Clues may be anywhere and need to be diligently sought. In the case of a woman, the history should include questioning about the cata menia which may aggravate congestive symptoms and signs and about the effect of childbirths How many preg nancies have there been? Were they all well tolerated? Are all the children living? How old are they? And does the mother have adequate help at home?

and since this is so and since some of these diseases start only in middle age or later it behooves us in all cases, young and old, to record a clear family history as to these diseases. As to how they or the susceptibility thereto are passed on we have only theories.

Rheumatic fever and rheumatic heart disease do often run in families and should always be asked about. Is the tendency due to a family allergy to the Streptococcus hemolyticus?

Hypertension (or the old Bright's disease) is often a family complaint. But some families are simply nervous hyperreactors and never get real hypertension. The combination of inherited hypertension and nervous hyperreactivity is especially unfortunate.

Coronary atherosclerosis of high degree causing angina pectoris, coronary thrombosis, myocardial infarction, or sudden death is notably commoner in the children of parents who give such a history and especially if both parents are so affected in youth or middle age. Is that due to familial hypercholesterolemia of greater or lesser degree, to excessive mesomorphic build which is certainly readily inherited, to an inherited inadequacy of a coronary arterial tree, to hormonal imbalances, to familial gluttony or to combinations of these factors or others not yet under fire?

Neurocirculatory asthenia and familial tendencies to develop worry about heart disease, whether such is present or not, should always be enquired about since they are commonly found in both parents and children and can account for much of the symptomatology. Happily they do not themselves predispose to heart disease or shorten life but they can make the lives of generations of people rather miserable. One of my patients with neurocirculatory asthenia who was discharged from military service in World War I because of disability due to that condition

had a son who was discharged from military service in World War II for the same reason.

Finally *unusual longevity* itself is a family trait and should be carefully enquired about. It is well known that a person who has recent ancestors parents or grandparents or both who have lived to be very old that is beyond 80 years, is likely to live to be old also and to survive illnesses, operations and accidents more readily than does another person whose parents died rather young of cardiovascular disease The outlook is especially favorable if there have been long lived ancestors on both sides of the family Years ago many young persons died of infections that are now happily wiped out or sharply controlled. Such deaths in our family history recording should be noted but largely discounted For example my father s mother died of cholera before he was a year old and his father died not very long afterwards, also quite young, of phthisis. But much more important than their early deaths as current clues was the survival of his father s mother into her hundredth year she was born in 1800 and she died in 1900 In prognosis especially is familial longevity a very important clue

In closing this chapter I should add a few words about the health especially that of the heart of the spouse. Worry over this may aggravate or precipitate trouble due to heart disease or there may be an anxiety syndrome due to the beginning of symptoms which may seem to mimic those that crippled or killed the husband or wife. Occasional patients, mostly women with neurocirculatory asthenia sometimes called anxiety neurosis have consulted me because they feared that they too had the heart disease generally coronary in type that had affected their husbands, not infrequently and dramatically causing their sudden deaths. Or the husband or wife already a cardiac patient

requests an examination t tl
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confusion about sympt ms t l
one of the married couple an l

CHAPTER 4

SYMPTOMS BREATHING DIFFICULTIES

More valuable cardiovascular clues in diagnosis and treatment come from subjective evidence of the present illness, that is from symptoms, than from any other category For this reason the most meticulous care should be directed to obtain the best possible history of the present illness with the details of the symptoms. More time is usually necessary for this part of the examination than for any other and it should be conducted by the physician himself or herself and not relegated to student, assistant secretary or nurse Experience enhances the value of the history but thoughtful consideration of the importance of this part of the examination will allow even a relative beginner to do a good job

Of all the symptoms the difficulties in breathing are the most diffuse and important in differential diagnosis and so they will be discussed first in the present chapter

Important cardiovascular clues are related to breathing, and yet the great majority of persons who have difficulties of respiration have no heart disease at all Bronchial asthma, infections of the lungs or bronchi pleurisy nervousness, poor physical fitness and even extreme obesity are common causes of dyspnea. Less common causes are pericarditis pneumothorax intrathoracic tumors and dis-

orders of the brain or of its circulation. These various conditions may be mistaken for heart failure and even treated wrongly as such particularly if they happen to be present in persons who happen to have some heart disease too. Especially is this true in the case of old men and old women with dyspnea and wheezing due to chronic bronchitis and emphysema with no heart trouble at all but because they are old they are given digitalis without benefit.

Clues which help to rule out heart disease as a cause of dyspnea are

1) X ray evidence of a small heart shadow not infrequently vertical in position with low poorly moving diaphragm in the case of pulmonary emphysema.

2) A normal electrocardiogram.

3) Wheezing induced by certain foods pollens or dusts and cleared by elimination of such factors or by epinephrine.

4) Asthmatic attacks which have recurred for many years

5) Sighing respiration with a tendency to hyperventilate along with multiple other symptoms in particular heart ache palpitation faintness, marked fatigability and nervousness in the case of neurocirculatory asthenia.

6) Pain on breathing deep or superficial whether or not one hears a pleural or pericardial friction rub in the case of pleurisy or pericarditis.

7) Fever tachycardia, and leukocytosis in the case of respiratory infections and pulmonary infarction

When heart disease is also present along with these clues it becomes doubly important to determine so far as possible whether the heart disease is partly responsible for dyspnea or whether it is but coincidental and silent. One important kind of heart disease that may be serious and yet not the cause of dyspnea is that due to coronary atherosclerotic narrowing. *An older man may have had angina pectoris for*

years but never myocardial infarction or other possible cause for cardiac enlargement or left ventricular failure leading to dyspnea therefore, some other than cardiac cause of the shortness of breath should be diligently sought. It is likely to be found as chronic bronchitis and emphysema.

In rare cases it is difficult to exonerate the heart completely and then it is not only permissible but in truth wise to test therapeutically If digitalis or a low sodium intake or mercurial diuresis, is quickly that is, within two or three days followed by relief of dyspnea, one may with reason in doubtful cases blame the heart for at least some of the difficult breathing if a coincidental improvement can be fairly well ruled out.

In the case of the sighing dyspnea of neurocirculatory asthenia hyperventilation may induce faintness and other symptoms which alarm the patient. It is helpful sometimes to demonstrate to such cases the relative unimportance of these symptoms by asking these patients to hyperventilate in the presence of the physician at the time of the first interview

The most distressing dyspnea of all which tends to come on very suddenly often waking a person up at night is that associated with pulmonary edema. There may or may not be wheezing with it if there is serious heart disease present such asthma is ordinarily called cardiac asthma. However even in the presence of serious heart disease pulmonary edema with its intense dyspnea and orthopnea may be due to something else most often to acute pulmonary embolism With no heart disease at all pulmonary edema with its distressing dyspnea can be excited by pulmonary embolism and now and then by other causes too, even a thoracentesis.

Clues which point to the diagnosis of acute pulmonary embolism should be always borne in mind in differential

diagnosis. A sudden attack of dyspnea with or without cough or asthmatic breathing especially in a patient with chronic heart disease and congestive failure or postoperatively attended by considerable tachycardia and often followed by slight to moderate fever and leukocytosis is not infrequently due to pulmonary embolism with infarction. There may be no fever unless there is infarction which is more likely to occur if there is already congestive heart failure and a faulty pulmonary circulation. There may or may not be hemoptysis copious or slight, with pulmonary infarction and there may or may not be signs of the acute cor pulmonale dependent on dilatation and anoxia of the right ventricle. The electrocardiographic evidence of the acute cor pulmonale which we have found in about 10 per cent of our cases of pulmonary embolism (the ones with the larger emboli as a rule) consists of the appearance or accentuation of S waves in Lead 1 and of Q waves in Lead 3, increased R waves in Leads V and V, and inverted T waves or an increase of their inversion in Leads 3, aVF, V_1, and V.

An important clue to the diagnosis of pulmonary embolism is recent unilateral swelling of a leg due to venous thrombosis or asymmetrical edema if both legs are swollen. Even equality of leg edema may be a clue if no other cause such as congestive failure is found for it. This clue should always be looked for but it is not infallible and there may be no swelling at all even in the presence of leg vein thrombosis, and of course, thrombosis and swelling may be present in the legs without embolism and with other cause for the acute dyspnea. The Homans sign (soreness in calf when the foot is flexed) is a useful clue also when it is present but, like tenderness on pressure over the calves themselves, it is not infallible.

One other rather uncomon clue to a pulmonary infarct *of large size in the presence of congestive heart failure with*

engorgement of the liver is jaundice due to inability to dispose quickly enough of the excessive blood pigment

Other noncardiac causes of abrupt or rapidly developing dyspnea include spontaneous pneumothorax, acute pleurisy or pericarditis (here the breathing is usually painful as well as difficult) and dissection or rupture of the aorta. Further consideration will be directed to these conditions in the chapter on Pain

Cardiac causes of dyspnea whether acute or chronic, are essentially two failure of the left ventricle and mitral stenosis, in the former due to *myocardial insufficiency* and in the latter to valvular stenosis or regurgitation or both that is a *mechanical factor*

The left ventricle is much more often primarily under strain pathologically than is the right ventricle and so every patient with chronic hypertension chronic aortic valvular disease or myocardial infarction (acute or chronic) due to coronary thrombosis should be carefully followed as to clues of beginning failure of the left ventricle these are dyspnea on ordinary effort or suddenly at rest, apical gallop rhythm accentuation of the pulmonary second sound, and alternation of the pulse If these clues appear treatment for heart failure should begin at once

Failure of the right ventricle does not cause dyspnea until it is far advanced at which time air space in the lungs is restricted by congestive hydrothorax or by a big liver and ascites raising the diaphragm except that a faulty cerebral circulation does favor Cheyne Stokes breathing or less marked periodicity and difficulty of respiration

Sudden dyspnea in mitral stenosis before the right ventricle fails may be due to extracardiac causation *e.g* pulmonary embolism but it is usually due to sudden pulmonary edema secondary to the narrowing of the mitral orifice which prevents the blood which is flooding the lungs from flowing fast enough through the heart.

Although sudden effort may cause the left ventricle to fail quickly or the lungs to be congested in the presence of mitral stenosis and although some abrupt complication like acute myocardial infarction or a pulmonary embolus may act similarly a common and important exciting factor precipitating either condition, and thereby acute pulmonary edema, is paroxysmal tachycardia, either atrial or ventricular or paroxysmal atrial fibrillation. A sudden rise of 60 to 80 beats as from 80 to 150 is often too great a strain for the limited reserve of either left ventricular myocardium or pulmonary circulation. Since usually measures are available to control such arrhythmias, or at least the ventricular rates, it is of the utmost importance to seek this clue as to heart rate by question or observation or electrocardiogram

The position of the heart in the body has an important influence on breathing The commonest descriptive phrase applicable to this is orthopnea which means literally breathing in the upright position This position is, of course assumed by the victim of dyspnea for comfort s sake It is found in a great variety of conditions and is not strictly diagnostic. It is almost always present whenever there is severe shortness of breath on effort or moderate shortness of breath at rest. It is, of course explained by the greater air space available for oxygenation of the blood in the upright position An interesting variant of orthopnea is the attitude assumed by certain individuals, especially those with pericardial effusion or very large hearts, who are most comfortable when leaning way forward often with chest resting on a few pillows or on a bed table. Some patients who are unable to breath comfortably lying on their backs or sides do find that they can breath with fair comfort if they lie face down It is well to remember this at the beginning to test its effect and even perhaps to use it therapeutically Finally the *squatting posit on assumed*

by children with congenital heart disease with the morbus caeruleus that is the cyanotic type of congenital heart dis ease is well known. It is probably helpful because the individual affected is at rest in the upright position. For children, fair comfort in this position is readily acquired but for adults this position is uncomfortable and sitting in a chair is more suitable

One other important type of dyspnea due more to cerebral vascular insufficiency than to heart failure itself is the periodic apneic and hyperneic respiration of Cheyne Stokes. A combination of cerebral vascular cardiac, and renal insufficiency is particularly likely to lead to Cheyne Stokes breathing It is caused by the alternating stimulus of CO excess in the blood and depression of oxygen satura tion and CO lack. This is often as distressing for the observer as it is for the patient because of the long periods of no breathing at all It tends to be worse at night and is aggravated by opiates or sedatives but helped especially by intravenous or rectal aminophyllin

Of all the technical methods those that give us the rich est clues in the elucidation of difficulties in breathing are *fluoroscopy* and *the x ray film* In the first place we can determine the presence or absence of serious pulmonary disease intrathoracic tumors, hydrothorax pneumothorax diffuse or localized pulmonary edema or infarcts, and emphysema And secondly we can study the heart as to its size shape position and action and the presence or absence of extensive pericardial effusions or calcification and the hilus shadows which represent the main pulmonary arteries and veins Certain clues are quickly derived such as the following the first two of which were mentioned earlier in the chapter

1) Dyspnea cannot be ascribed to myocardial failure if the heart shadow is small or normal in size

2) A very low diaphragm which moves little with res-

piration is a sign of emphysema which can itself cause dyspnea whether the heart is diseased or not

3) Excessive fulness of pulmonary arc and lung hilus shadows in the absence of congenital defects, in particular atrial septal defect and patent ductus arteriosus is evidence in favor of the diagnosis of left ventricular failure or of mitral stenosis as a cause of dyspnea. The differentiation between these two causes should be easy through physical examination and electrocardiography

4) Fluid limited to or preponderantly in the left side of the thorax indicates the probability of some factor other than heart failure (e.g pulmonary infarction or infection) as a cause of dyspnea.

Measurement of the vital capacity is of interest in comparing the breathing capacity on different occasions in the same individual but it is not diagnostic One may have sharp limitation of the vital capacity when there is disease of the lungs, when there is heart failure when there is fluid or air in the chest, and when there is neurocirculatory asthenia Determination of the blood gases and blood pressure in various parts of the circulation is also of general interest, again not very helpful in differential diagnosis except in the intricacies of the elucidation of the particular *defects in congenital heart disease. Here the data obtained* by cardiac catheterization are of much value and the reader is referred for discussion of this information to larger works that deal with details of analysis of congenital and acquired cardiac defects.

Thus, it is evident that despite the complexity of this symptom of difficulty of breathing there are many clues to help us decide the degree of responsibility of the heart in its production and to indicate the proper treatment

CHAPTER 5

PAIN

ONE OF the most important and intriguing symptoms of cardiovascular interest is pain Sometimes and in fact in the great majority of cases this symptom is easy to interpret but often it can be confusing Its proper analysis is of the greatest importance in differential diagnosis. At the very outset it should be recognized that one of the reasons for confusion is that there are in many cases not just one pain which is thought to be caused by one condition or another but actually two or three different pains, often closely resembling each other in position or character or circumstance but each indicative of some special disease or disorder It is also very important to recognize that one of these pains may stir up another as an aggravating but not causative factor The extra time needed for adequate query or test to untangle complicated problems related to pain is time well spent on the occasion of the first examination of a patient and may save weeks or months of uncertainty subjection to much needless investigation by x ray and other procedures, and useless therapy

The definition of pain is actually very broad and is variously interpreted by different individuals so that if necessary one should go into some details about definitions with the patient at the very beginning Some persons call almost any discomfort pain or at least painful while others limit

their interpretation of pain to a sharp or cutting sensation while a dull discomfort is called an ache or a soreness or a *misery or a hurt. A good many of my patients with typical* angina pectoris have refused to call it a pain but have instead called it a pressure, a choking (which is the literal translation of the Greek word angina) a heaviness or even a difficulty in breathing when actually there is no disorder of respiration per se If a patient complains of difficulty or heaviness in breathing for which there is no apparent reason, it is well to ask him where this difficulty is. If he places his hand over the sternum one should suspect that angina pectoris is responsible for the symptom which he interprets as a breathing difficulty Since this is a fairly common experience it behooves the doctor to take plenty of time in obtaining the patient's history if there seems to be any question about the presence or absence of pain.

There is one other very important basic consideration and that concerns the sensibility or nervous sensitiveness of the patient. It is, of course an axiom widely quoted that in the practice of medicine the host must be considered equally with the disease or injury itself This is particularly true of the symptoms of cardiovascular disease. A relatively insensitive person may be hard hit before he feels sick while a very sensitive patient may feel badly with relatively little disease present. A corollary of this is that a person, no matter how sensitive, can school himself or herself to endure pain or other symptoms with overmuch fortitude hiding his or her illness from family and friends and, indeed, even from the doctor until it may have become far advanced This matter of sensitiveness and endurance must be carefully weighed by the physician at the time of the first examination. The attitude of the patient while he tells the story his presentation of facts, the attitude and comments of whatever member of the family is present or near

at hand and a few findings on physical examination are useful clues For example, the patient may be obviously very nervous or apprehensive may sigh or tremble may perspire or show a fast heart rate at the time, his reflexes especially the knee jerks, may be hyperactive and he may obviously exaggerate or indeed minimize his pain or other symptoms All this reaction is fully as important in diagnosis and treatment as the details of the symptoms themselves. Incidentally a point of much significance sometimes over looked is that a very nervously sensitive person can also become very ill with coronary heart disease for example but at first the disease itself may be underestimated or even ignored by family and doctor because of the fact that the person in question has always been nervous and apprehensive and indeed not infrequently hypochondriacal One must be on the outlook for this.

Coronary Insufficiency Of all the pains and disagreeable sensations caused by heart trouble or related directly with the heart that due to coronary insufficiency is the most important and so will be discussed first It should, however be observed that coronary insufficiency can occur without pain Although this is true this fact has on occasion been overemphasized There are to be sure some patients who fail to present subjective evidence of acute or subacute coronary insufficiency which is clearly evident by objective findings but these cases are relatively very few in number In them there is either a masking of the pain by the presence of a state of shock as for example in acute coronary thrombosis or postoperatively or by anesthesia induced either for surgery or other therapy or indeed even via alcohol or by a psychotic state or extensive disease of the central nervous system which can make interpretation of symptoms difficult or impossible or finally and least commonly of all by an extraordinary lack of sensibility It

has been said that a full blooded negro does not have the characteristic symptom of angina pectoris in the presence of coronary insufficiency We need however much more accurate information than we have as yet to know the *degree of correctness of this observation. At any rate the* chief reason as I have found for the statement that painless coronary insufficiency is relatively common is the failure of observers to obtain adequate histories of the patients concerned.

In the great majority of my patients the diagnosis of coronary insufficiency is easy based on the presence of the characteristic symptom of angina pectoris. I would estimate that in at least 90 per cent of patients there is no difficulty whatsoever and in them one doesn t need to seek for other clues

The typical story is of course that of substernal oppression high low or intermediate in position with or without radiation of a numbness to both forearms but usually preponderantly in or even limited to the left side lasting a few minutes at a time induced characteristically by effort or excitement, and relieved by rest or nitroglycerine. *The pain doesn t need to be severe* as a matter of fact it is often mild and sometimes only a suspicion of a pain In my experience it is rarely attended by the sense of impending doom or dissolution so often mentioned by past observers such apprehension of death I have found actually more often in nervous persons without angina pectoris but with such a condition as neurocirculatory asthenia. A helpful clue to the diagnosis is the gesture of the patient in locating the position of the pain he practically never points to it with one finger but places a palm or even both palms over the front of the chest The palm is usually laid over the sternum in the case of angina pectoris If it is placed over the left breast the ache of neurocirculatory asthenia is more

likely to be the answer As already stated, one should remember that many patients refuse to call the sensation pain but use other terms to designate the heaviness or choking that they feel Heberden himself in coining the words angina pectoris in 1768 picked out the Greek word for strangling to distinguish it from other kinds of pain or *dolor*

One of the most interesting and important variants of angina pectoris or of pain due to coronary insufficiency is the discomfort that may be induced thereby outside the chest altogether or in the back Usually the pain starts characteristically substernally and then radiates to or is followed or accompanied by pain in left arm wrist or hand most commonly right arm sometimes and neck jaws teeth back or upper abdomen less often. Radiation of substernal pain to the jaws or teeth strongly favors its interpretation as angina pectoris I have never observed its radiation or occurrence in the lower abdomen or legs, but I have encountered it in the flanks. On occasion the radiation may be reversed in its direction starting outside the chest but radiating finally to the substernal area In very rare instances the pain may be actually limited to one or more of these outlying points and in such cases the diagnosis may be very difficult It should be added that radiation of pain to the left arm does not establish the diagnosis of coronary insufficiency since there are other causes

The most important clues are the production of the pain by walking fast or uphill without undue motion of the part affected such as wrist arm shoulder or neck and by its quick relief by nitroglycerine Slowing the heart rate by carotid sinus pressure has also been noted to clear the pain away more quickly If the pain is produced only on local motion and not by walking fast one cannot ascribe it to coronary insufficiency The final clue to this unusual mani

festation of coronary insufficiency is the presence of some previous or current injury to the area that is painful or other lesion of the part for example, sprain or strain, displaced intervertebral disc, arthritis bursitis or severely infected tooth root. Thus such a pain position might be ascribed to a sympathetic sensory distribution or radiation.

Two other observations about angina pectoris are of special interest. One is that this symptom is commonest at the beginning of the day (for example on walking to garage, bus or train after breakfast) and at the beginning of some exercise such as golf, a physiological vasodilatation affording protection later The other is that in the case of more serious coronary insufficiency angina pectoris may occur on first lying down at night or later to awaken the patient this is called *angina pectoris decubitus* and demands respectful consideration and treatment and careful differentiation from the pain induced by a hiatus hernia which does not cause pain on walking but is often relieved thereby

Clues that are helpful in ruling out angina pectoris are numerous. Among them are the following 1) a sharp stabbing or needle like character of the pain, 2) a throbbing pain 3) a very short duration of a few seconds only 4) a very long duration of a half hour or more unless coronary thrombosis or some continuing strain such as continuous effort, excitement or tachycardia (especially paroxysmal in type) is in progress, 5) induction of the discomfort by eating or during rest seated and not by effort in the same case or recumbency in the supine position 6) relief of chest pain by effort, 7) induction of the pain by light or moderate use of the arms especially by raising an arm without any discomfort during fast walking 8) induction or aggravation of chest pain by deep breathing, and 9) tenderness over the site of the pain.

I would add at this point that the particular causes responsible for the clues just mentioned are in particular conditions such as a) digestive disorders, especially spasm of esophagus or cardia of the stomach (cardiospasm) that may not only mimic angina pectoris due to coronary insufficiency but not uncommonly complicate it b) bruises, sprains, arthritis bursitis, and other such abnormalities in chest wall spine shoulders and neck, c) neurocirculatory asthenia, d) neuritis as in herpes zoster and e) pericarditis Skillful and experienced questioning and physical examining usually suffice for the unravelling and proper application of the various clues even before resorting to laboratory procedures in particular of course to electrocardiography

The commonest mistakes are of commission rather than of omission and in calling other troubles coronary insufficiency Cardiospasm, caused by irritability of esophagus and stomach often produces substernal discomfort that simulates angina pectoris in position character and duration but not in the circumstances of its occurrence The pains are apparently not however exactly alike since a good many patients who have both can tell them apart The digestive disorder often extends over much of the patient's lifetime although in varying degree it may decrease or even disappear when smoking is stopped or nervous strains subside. Sometimes the discomfort caused by cardiospasm ends in the belching of gas and when an attack of angina pectoris is accompanied by cardiospasm or is a cause thereof the coronary insufficiency pain may seem to be unusually prolonged and to be relieved by a belch

Pain from a skeletal abnormality of some kind in bones, joints, muscles, and bursae can be very confusing especially if the pain therefrom is substernal or in the left shoulder or arm There are several clues to such an origin of pain As already noted above motion of the affected part usually

induces pain, as may certain positions in bed at night. Tenderness on pressure over the site of the pain is strong evidence in support of this diagnosis since that sign is not an accompaniment of uncomplicated angina pectoris. There may be a stiffness of a joint or of the whole neck or of an arm. An important query concerns the origin of the lesion there may have been an accident or a muscle strain, arthritis may have begun to appear in other parts of the body or there may have been prolonged inactivity as in the case of bed rest for various ills such as myocardial infarction. The so-called shoulder arm syndrome may arise in this way complicating the convalescence of some patients after coronary thrombosis but it has been a rare complication in my coronary heart disease cases and is not, *in my opinion to be related any more to the coronary* disease itself than to any other cause of long invalidism perhaps my long established custom of relatively short bed rest (not over three weeks) has helped to keep down such a condition

Neurocirculatory asthenia is often wrongly interpreted as coronary insufficiency just as it is also wrongly treated as myocardial insufficiency The very multiplicity of the symptoms of this condition causes confusion even though it is perhaps the chief clue to the correct diagnosis. Here the prolonged heartache often accompanied by precordial tenderness on percussion or palpation is very different from angina pectoris. Hasty questioning may however miss this easy differentiation as to position, character and duration since the patient simply volunteers the information that he has anterior chest pain induced or aggravated by effort or excitement This one common bond with angina pectoris is no reason for misdiagnosis but its prominence in the over all picture of neurocirculatory asthenia was responsible for its early label as the effort syndrome

In most cases of neurocirculatory asthenia the chest pain is less complained of than is the difficulty of breathing of the sighing type which was discussed in the chapter before this one

True neuritis involving chest or arms is far down the list in differential diagnosis. Pressure on nerve roots toxic neuritis and inflammatory disease give rise as a rule to symptoms easily distinguished from angina pectoris. The pain is rarely substernal it is sharp and more like a tooth ache and is of long duration although it may come and go rapidly as a throbbing sensation The very disagreeable left chest pain of herpes zoster may simulate the pain of coronary thrombosis in duration and severity but its posi tion is different in that it extends much further posteriorly often follows a narrower bandlike course and reveals no abnormality in the electrocardiogram Also it is a very much rarer condition and when it is responsible for an obscure chest pain reveals itself eventually in the crop of herpetic skin lesions which should always be looked for in the follow up of a diagnostic problem Finally the term intercostal neuralgia should be applied with great hesi tation if ever It covers a multitude of conditions some of which like muscle strain can be readily diagnosed with *a little care Generally the term means nothing except an* admission of ignorance which is better expressed in some other way such as chest pain of unknown origin

Earlier in this chapter I have referred to the pain of *acute coronary thrombosis* due to myocardial anoxia or infarction as well as to the pain of angina pectoris. As a matter of fact, they have exactly the same characteristics except for the long duration of the former and its greater tendency to cause collapse Very rarely is there prostration with angina pectoris *although there is such a condition as* that described in 1799 by Parry which he labelled syncope

anginosa. Very infrequently a patient may actually faint with angina pectoris, apparently through a sudden abrupt *drop in pressure* and incidentally *it is possible to induce* syncope in a susceptible person by an average dose of nitroglycerine (1/100 grain or 0.0006 gram). It is on the other hand quite common for a patient suffering from acute coronary *thrombosis* to develop a *sharp drop in* blood pressure and a state of shock. The reverse is also possible in fewer cases of acute coronary thrombosis, that is, a rise of blood pressure to *high* levels temporarily during the pain which apparently acts as a pressor agent.

Reference should here be made to clues to distinguish three severe fulminating accidents to health from the condition of *acute coronary thrombosis which they sometimes* resemble. The first of these is quite common and consists of *pulmonary embolism*. Although there may be precordial pain with such an episode, especially when there is an inadequate *coronary circulation to start with*, there are distinguishing features which consist as a rule of a sharp increase in heart and respiratory rates as well as fever when there is embolism, lung signs including localized rales or consolidation, x-ray evidence of atelectasis or scarring in lungs, in a few cases of the acute cor pulmonale characteristic changes in the electrocardiogram, and later pleurisy pain and a friction rub and sometimes tenderness over the site of a pulmonary infarct. As a matter of fact both conditions may occur together, pulmonary embolism complicating acute myocardial infarction and vice versa.

The second condition to be differentiated is an *acute abdominal emergency* such as the passage or obstruction of a gall stone, rupture of a viscus (*e.g.* through a peptic ulcer), mesenteric *thrombosis or embolism*, or a rupture of an arteriosclerotic aneurysm. Since the symptoms and signs of these conditions are almost all subdiaphragmatic,

an error should rarely be made On occasion however we need all tests including electrocardiography to be sure and again we must remember that both conditions are often associated (without causal relationship) This is especially true of gallbladder disease and myocardial infarction It should be noted that although there is sometimes coronary insufficiency pain limited to an arm which therefore might be called angina brachialis there is no real entity such as angina abdominis due to coronary insufficiency if one excludes pain located in the epigastrium which radiates neither up nor down Sometimes an arteriosclerotic aneurysm in the abdomen may ache and a marked sclerotic or thrombotic narrowing of the mesenteric arteries with severe reduction of blood supply to the intestines can cause severe pain due to anoxia which has sometimes been called angina abdominis. Further evolution of this process may lead to intestinal infarction with or without complete thrombotic mesenteric occlusion

Finally there is *aortic wall dissection* in the chest The abrupt onset of excruciating pain often maximal at the start its radiation as a rule to the back and often down the spine to the legs the involvement of various aortic branches with interference of the blood flow therein the absence of electrocardiographic changes unless the coronary flow is interfered with by hemorrhage in the coronary artery walls and the much higher early mortality in the case of the dissecting aneurysm of the aorta are all clues pointing to the aortic lesion.

Next, we come to inflammatory lesions in the thorax in particular *pleuritis* and *pericarditis*. The former is common when uncombined with the latter and gives rise to pain that is as a rule not precordial but in the lateral or posterior portions of the chest. Acute pericarditis however does often give rise to precordial pain which is sometimes

severe and not infrequently associated with pleuritis (pleuropericarditis) Such pain, attended by fever and leukocytosis and by serial changes in the electrocardiogram can simulate acute myocardial infarction and needs careful evaluation A helpful clue is the increase of the pain during full deep respiration in the case of infectious pericarditis. Such aggravation by breathing is rare in acute myocardial infarction even when there is a pericardial friction rub due to involvement of the epicardium by the infarct. There are exceptions in cases of infarction with extensive pericarditis when the parietal pericardium and adjacent pleura are also invol ed. Another clue is the early appearance of fever and friction rub immediately after the pain of pericarditis begins or indeed occurring coincidentally Although the electrocardiogram is abnormal in most instances of acute pericarditis the pattern can be as a rule easily distinguished from that of acute myocardial infarction.

Up to now in this chapter the discussion of cardiovascular pain has been limited to that dependent on coronary insufficiency or thrombosis to pericarditis, and to dissection of the aortic wall and as a matter of fact there is little else to present of importance concerning pain in the l eart and great essels

Pa n due to pressure from or rupture of an aortic an urysm in the thorax is far less common nowadays than it was a generation ago because of the sharp decrease of aortic syphilis. It can usually be easily identified by its character duration and especially of course by x ray confirmation of the presence of the aneurysm The heartache of neurocirc ulatory a thenia has alrea ly been referred to it is common and readily diagnosed on the basis of its character of the type of person affected and of the coincident symptoms which always accompany it Pulmonary vascular

pain has been occasionally referred to as a basis for the so-called *angina hypercyanotica* Sudden stretching of the pulmonary artery walls or of those of its main branches does occur acutely when there is a massive pulmonary embolus and may perhaps account for some of the discomfort which accompanies the extreme dyspnea of such an episode but in such cases coronary insufficiency at least in part due to myocardial anoxia or hypoxia in an older person with limited coronary reserve can also cause distress at such a time Pressure from a rare pulmonary artery aneurysm is a very rare cause of chest pain

In closing this chapter an important basis for typical angina pectoris not due to coronary atherosclerosis or obstruction should be mentioned. It is possible to have a heartache or almost uniquely even angina pectoris when there is *myocardial disease* due to rheumatic fever or other cause Very recently I encountered a case of so-called endocardial or endomyocardial fibroelastosis with very heavy heart whose case I had followed for 15 years before he died at the age of 71 of congestive failure. He had had absolutely characteristic angina pectoris on effort during all those years and had slowly developed left bundle branch block but at autopsy his coronary arteries were capacious, there was very little coronary atherosclerosis and the myocardium showed no infarction In some cases there may be to be sure, some coronary narrowing but far too little to explain the pain There is one thing in common in these cases and in many of them a subsidiary factor The explanation is probably to be found in the extra load imposed on a myocardium which is not strong enough to support the load with the available oxygen supplied by a coronary circulation which should be adequate for a stronger muscle or for a lesser load Thus one finds left ventricular enlargement or even total ventricular hyper

trophy insufficient to carry the burden of severe hyper tension of large myocardial scarring, or of aortic valve disease (stenosis or regurgitation) without more oxygen. That happens before the myocardium actually fails with ensuing congestion in lungs or systemi circulation.

Once myocardial insufficiency sets in, the symptomatic evidence of coronary insufficiency subsides although at the turn both may be present even from day to day This particular sequence of events is true whether the angina pectoris is due to coronary arterial obstruction or to the much less common mechanism described above

The subsidiary factor mentioned is the effect of a highly sensitive nervous system which reacts suddenly to increase the heart load by raising heart rate and blood pressure on excitement or effort. And there is one final factor behind angina pectoris in some cases of little or no coronary ar terial narrowing and that is a high degree of *aortic valve stenosis* usually acquired and rheumatic in origin, which sharply reduces the stroke output of the heart and thereby limits the amount of blood which enters the coronary arteries An important cause of sudden death is high grade aortic stenosis.

CHAPTER 6

PALPITATION

PALPITATION *is the least important of the* three common cardiac symptoms and yet it is often of great significance as a clue to explain the sudden onset of either of the other two dyspnea and pain or to reveal the cause of a cardiac neurosis.

By definition palpitation is the consciousness usually disagreeable of the heart beat whether regular or irregular in rhythm and whether fast slow or of average rate It occurs whether the heart is diseased or not and is in major degree dependent on the nervous sensibility of the person under consideration. Thus a very sensitive individual may feel disagreeably even a relatively slight increase of the normal heart rate (*sinus tachycardia*) he may wince at or be terrified by a completely unimportant extrasystole or premature contraction or he may be completely prostrated by a paroxysm of tachycardia which although annoying, may be quite benign On the other hand an insensitive person may be quite unconscious of the occurrence of extrasystoles and even hardly aware of considerable tachycardia whether paroxysmal or not. Those are of course the extremes most individuals fall into the large intermediate group

A further basic consideration concerns the findings at the time of examination of a person who complains of

palpitation. The heart may then be regular and well within the normal range of rate, and yet the past or recent history of the patient may clearly reveal a disorder of rhythm at the time of some episode of chest pain, shortness of breath faintness or malaise lasting seconds, minutes, hours days or even weeks Too hasty a questioning may fail to uncover this important clue and the patient may be wrongly regarded as wholly neurotic or as having suffered from serious heart attacks neither of which extreme appraisals is correct. Two illustrative cases which I encountered during the second World War are examples of a common failure to look for important clues or to interpret them properly when they are found Both of these cases were hypersensitive young soldiers with cardiac neuroses but no heart disease Both had had paroxysms of tachycardia. In one case the doctor who made an examination a few hours after a paroxysm found nothing wrong and overdid his reassurance intimating that the heart attack was an imaginary one this attitude caused an antagonism and distrust which deepened a cardiac neurosis already beginning Equally unfortunate was the attitude of a physician who saw the second young man during a paroxysm of tachycardia prolonged bed rest and various medicines were prescribed and continued for sometime after the paroxysm was over and the patient was left to believe that he had had a serious well nigh fatal heart attack establishing a cardiac neurosis of high degree. Some years ago an especially unfortunate case of this sort came under my observation. A middle-aged woman otherwise in good health was admitted to the hospital for an addiction to morphine that had extended for many years following the overtreatment of a few paroxysms of tachycardia in her youth. There was no evidence whatsoever of any heart disease or other illness except for the morphinism and yet she

had acquired such a sensitivity to an increase of her heart rate of only 10 to 20 beats as on the occasion of my own examination of her that she would at such times cry out in anguish for more of her soothing medicine With some difficulty she was restored to good health with a cure of her opiate addiction.

The commonest type of palpitation is simply consciousness of a forceful heart beat during normal rhythm whether the rate is rapid, slow or average There are many causes, including effort, excitement, fever toxic states, and trauma. Rarely however is this symptom itself bothersome enough to be a specific complaint.

Next in frequency are *extrasystoles* or *premature contractions* The great majority of persons who show extrasystoles have no heart disease Such beats are commoner in cardiac patients than in noncardiacs and also they are more numerous with increasing age Most persons are conscious of this disorder of rhythm when it first occurs but they often become accustomed to it and after a while may not notice it at all especially if it comes regularly every few beats and indeed even as often as every other beat The arrhythmia may be felt as the early beat itself or as the pause following the premature beat or as the post-extrasystolic thump sometimes all three sensations are felt I myself have noted a full sensation in the neck at about the time of a premature beat due doubtless to the wave of blood sent up from the atrium when it contracts at the time that the ventricle itself is still in contraction and the tricuspid valve is still closed. A few hypersensitive persons may feel extrasystoles as painful sensations and be fearful that they mean angina pectoris or otherwise indicate heart disease but reassurance relieves their anxiety although the disagreeable sensations may continue. In a case of very easily induced angina pectoris either the extrasystole itself

or the forceful post extrasystolic beat may be felt as a twinge of pain closely resembling angina pectoris of effort in character and location but not in duration. There is little or no difference in the sensations of atrial premature beats as compared with ventricular premature beats which are much more common Premature beats induced by exercise are more likely to be found in heart disease than are such beats occurring at rest.

The three more common abnormal mechanisms of the heart beat responsible for paroxysmal tachycardia are as follows atrial paroxysmal tachycardia, atrial fibrillation, and atrial flutter Clinical clues to distinguish between them may be helpful but, of course the electrocardiogram is the best technic of all in their differential diagnosis. *Atrial paroxysmal tachycardia* is the commonest and is often distinguished by its brevity lasting frequently only a minute or two up to an hour or two *it starts abruptly* (a circumstance often well described by the victim) and ends as suddenly although its termination may not be so well noted by the patient if sinus tachycardia follows it. It may on occasion, be stopped abruptly by carotid sinus pressure and it is almost invariably perfectly regular at rates between 130 and 180 per minute It most frequently occurs in persons with no heart disease.

Atrial fibrillation is a real arrhythmia with gross irregularity of rate which ranges in rate as a rule when untreated between 130 and 180 it lasts generally for hours, days, weeks, or often permanently and is not stopped by any measures except specific drugs in particular quinidine the heart rate in atrial fibrillation is however generally readily controlled by digitalis, in the absence of thyrotoxicosis, infection, or infarction but enough digitalis needs to be given to control the rate reasonably during mild exercise as well as at rest. Atrial fibrillation is much more com

mon in patients with heart disease than in persons without it is most frequent as a complication of mitral stenosis but it occurs in many other conditions, for example paroxysmally with pulmonary embolism.

Atrial flutter is more or less betwixt and between these other two atrial arrhythmias but like the others it starts suddenly it is usually attended by a regular rhythm and a rate of 150 the atria beating at twice that speed but able as a rule to transmit their stimulus to the ventricles only every other beat because of two to one heart block. On occasion spontaneously or as the result of vagal action via carotid sinus pressure or digitalis effect the degree of block may be increased regularly or irregularly so that only every fourth stimulus is transmitted or alternately third fourth fifth or sixth When the block is thus increased consistently the tachycardia is controlled although the actual atrial flutter mechanism may continue indefinitely Atrial flutter is less common than either fibrillation or paroxysmal tachycardia it lasts as a rule for days but it may be brief as for hours, or prolonged even for years.

A fourth type of paroxysmal tachycardia is that which originates in the ventricles (*ventricular paroxysmal tachycardia*) but it is much rarer and often much more serious prognostically than the other three types since it is more often associated with important heart disease particularly coronary in nature It is usually rather brief lasting minutes to hours, but like the other kinds of paroxysmal tachycardia it may precipitate coronary or myocardial failure in a cardiac patient its rate ranges usually from 140 to 180 and tends to be slightly irregular although generally an electrocardiogram is necessary to differentiate it from atrial paroxysmal tachycardia The differentiation is of importance because of differences in therapy For example in the case of the ventricular arrhythmias digitalis is out

of order in fact digitalis intoxication is one of the causes of this disorder of rhythm quinidine and pronestyl are frequently effective.

One other very rare type of paroxysmal tachycardia is that arising in the atrioventricular node it is called *nodal tachycardia* but can be distinguished only by electrocardiogram from atrial paroxysmal tachycardia which it resembles clinically

In contrast to the patients with cardiac neurosis precipitated by palpitation or by the wrong treatment thereof and to those with premature contractions and paroxysmal tachycardia without heart disease there are the very important and often serious cases of heart disease whose pain or dyspnea or even death may be caused by disorders of heart rhythm which occur usually as tachycardia regular or irregular and atrial or ventricular Quite often such patients do not complain and indeed may not even be aware, of the disturbance of rhythm itself because of the abrupt sequence of the vastly more disagreeable pain or dyspnea which masks it. Since, however it is of the greatest importance for the physician in diagnosis, prognosis and treatment to distinguish between coronary or myocardial insufficiency induced by overwork caused by effort, excitement or paroxysmal tachycardia of any type on the one hand and such insufficiency occurring essentially at rest on the other clues as to disorders of rhythm should be scrupulously looked for The paroxysm may be over by the time the patient is seen by the physician and therefore careful inquiry should be made as to the details of the patient's sensations at the onset or just before the onset of the pain or shortness of breath. Was there a racing of the heart first or simultaneously? Or if the heart did seem to beat fast during the attack did that follow after a bit the beginning of the pain or dyspnea itself and so seem to be a result

rather than a cause? And just how fast was the heart beat? Usually the patient cannot tell the heart rate with any degree of accuracy but he may recognize that it was about double the usual rate Sometimes it is impossible to answer this question at all and then it is wise to ask the patient to call the doctor at once at the onset of the next heart attack. The physician can then tell by observation or better still by electrocardiogram whether or not there is a dis order of rhythm. At times even on the first occasion the doctor may note an arrhythmia of which the patient is not conscious.

One of the most important clues in cardiovascular diag nosis and treatment is that of tachycardia whether atrial or ventricular responsible for prolonged angina pectoris or pulmonary edema In a person with much limitation of coronary reserve a *status anginosus* may be so induced and continued for hours, indeed so long that there is a close resemblance of the attack to one of acute coronary throm bosis which it is of the greatest importance to rule out. In the case of the status anginosus due to an abnormal tachy cardia the treatment primarily consists, of course in the treatment of the arrhythmia, to be followed usually by only a few hours of rest instead of weeks of rest as in the case of acute myocardial infarction. If the pain is severe during paroxysmal tachycardia morphine or other opium derivative may be necessary as in the case of coronary thrombosis but one does not need of course, to begin anti coagulant therapy Although the electrocardiogram reveals the abnormal mechanism it may not be very helpful other wise except as taken serially since the temporary myo cardial anoxia may cause temporary changes in *S-T* seg ments and *T* waves that cannot always be distinguished from the beginning changes of acute myocardial infarction. So soon as the tachycardia is over the electrocardiogram in

the arrhythmia group tends quite quickly (in hours or days) to return to its former state and, of course, the fever and leukocytosis of myocardial infarction are absent.

Much the same is true in the case of the paroxysmal dyspnea and pulmonary edema induced by paroxysmal tachycardia in two types of heart disease. One of these types is that of left ventricular myocardial weakness with low reserve secondary to hypertension aortic valve disease (stenosis, regurgitation, or both) or infarction (old or new) The newly imposed tachycardia proves to be too much of a burden the left ventricle fails and the lungs become congested. This state of affairs usually lasts for hours and subsides slowly after the tachycardia has cleared up which may happen spontaneously or by treatment via quinidine, pronestyl digitalis, or other specific drug or measure It may be simulated by acute massive or even moderate-sized pulmonary embolism which usually induces a sinus tachycardia of high degree even up to 140 to 160 per minute, thus resembling the heart rate in paroxysmal tachycardia It is obviously important to distinguish between these two conditions some of the clues have been presented in Chapter 4 on Breathing Difficulties but it is obvious that electrocardiography quickly distinguishes between the sinus tachycardia of pulmonary embolism and paroxysmal tachycardia as such except in the rare cases in which pulmonary embolism itself may actually induce a paroxysm. In any case no matter what causes the pulmonary edema morphine is the drug of choice in addition to any other specific measures such as quinidine and digitalis that are indicated.

The other very important basis for acute pulmonary edema induced by paroxysmal tachycardia is mitral stenosis. Here, of course, it is not myocardial weakness and failure that are the underlying cause but mechanical obstruction

which prevents a free flow through the lungs and left heart chambers of the blood that is pumped on vigorously by the till then unobstructed rapidly beating right ventricle Mitral valve surgery may solve this problem after careful study following the paroxysm.

Finally there is one other kind of palpitation due to *a v heart block* not associated with rapid atrial rate There may be true *dropped beats* as the result of which there is often consciousness of long pauses in the heart action followed by unusually forceful beats due to the expulsion of a larger amount of blood from the ventricles than usual this blood having accumulated during the longer pauses. Such pauses and hard thumps may occur after every second beat when the degree of block is three to two after every third beat when the grade is four to three after every fourth when five to four and so on. Or the grade of block may be very variable When the rhythm is regular though slow say at 40 due to two to one block or even slower at about 32 when there is *complete block* with independent ventricular action there is usually no palpitation Indeed some relatively insensitive persons are not even conscious of the arrhythmia in partial block If there is an interval of prolonged cardiac standstill in very high grade block, faintness and even syncope (*Adams Stokes syndrome*) may ensue this will be discussed in the next chapter

A very rare variant of the consciousness of an irregular bradycardia in a v block is that due to *s-a block* that is, an irregular slow discharge of impulses from the s-a node itself. It is to be differentiated from a v block by graphic records, most readily electrocardiographic It may occur in the so-called carotid sinus syndrome or other rare vagal or toxic depression of the sinoatrial pacemaker and is not prognostically so important as atrioventricular block

Bundle branch block produces no symptoms per se

CHAPTER 7

OTHER SYMPTOMS

VALUABLE clues to cardiac diagnosis and treatment are sometimes revealed by symptoms not so customarily related to the heart as are the three that have been discussed in the last three chapters, namely dyspnea, pain, and palpitation. Occasionally attention is called to them in individual papers but it will be helpful to present the more important ones together in the present chapter.

Faintness and actual syncope are common symptoms in general; only rarely are they due to heart trouble. However, on occasion they are very important and in the absence of other obvious cause should be investigated as a possible result of cerebral anoxia due to an abnormal heart rhythm or to faulty cerebral circulation caused by cerebral arteriosclerosis with some superimposed strain. There are five disorders of rhythm that may cause these symptoms. The commonest is an *extremely fast heart rate due to atrial paroxysmal tachycardia* which may be in the form of an ectopic atrial rhythm, either the usual type or atrial flutter (with 1 to 1 rhythm, for example). Much less common as a cause is the second type, namely an *ectopic ventricular rhythm*. Atrial fibrillation rarely induces a rate fast enough (that is, over 180 or 200 per minute) to cause even any pronounced faintness aside from actual syncope. As a matter of fact, loss of consciousness is rare with any type

of tachycardia though it does occur Once in a great while faintness or even actual syncope may occur briefly at the end of a paroxysm of tachycardia when the heart stands still just before the normal rhythm is resumed.

Syncope itself when of cardiac origin is more commonly due to a third disorder namely ventricular standstill than to any other cause such as extreme tachycardia. The most common type of ventricular standstill is that due to *very high grade partial heart block* in which no atrial stimulus passes the a v node and bundle for an appreciable length of time (minimum of about six to eight seconds) and in which there is no ready occurrence of ventricular escape

A vagal reflex, as from carotid sinus pressure may precipitate partial heart block with or without weakness and syncope Because of this possibility it is often wise to try to block this vagal reflex by belladonna or atropin itself which may tide over the worst of the symptoms. It is to be noted that ventricular standstill long enough to cause syncope may occur paroxysmally and to the surprise of the observer even though the electrocardiogram may show no a v block at all between syncopal attacks Another point of considerable importance to remember is that some persons with paroxysmal a v block which may induce syncope can faint away with cardiac standstill upon the application of pressure over the carotid sinus. The carotid sinus may not be unduly sensitive but the normal reflex may uncover the a v block and be wrongly diagnosed as an example of the carotid sinus syndrome Electrocardiographic study quickly corrects this error Inasmuch as any syncopal attack in the Adams-Stokes disease can be fatal it is wise to use as a test carotid sinus pressure only with great caution in an older person who may have either actual or potential a v block

The fourth disorder of rhythm which can cause faintness or syncope is *standstill of the whole heart* that is high

grade *sinoatrial block* long enough to induce paroxysmal atrial paralysis. This is very rare. Although it can be diagnosed by careful auscultation at the left sternal border to pick up the atrial contraction sounds or by careful observation of the jugular pulse with the subject lying supine (no waves are noted at all during the cardiac standstill in contrast to the regularly recurring a or atrial waves that are seen in cases of a v block) electrocardiography is the technic of choice to establish this diagnosis A hypersensitive carotid sinus resulting in the carotid sinus syndrome belongs here. It is in these rare cases that atropine is likely to be more helpful than in the patients with a v block.

The fifth and last disorder of rhythm that may cause syncope and that is easily confused with the true ventricular standstill of heart block is transient or paroxysmal *ventricular fibrillation* As a rule the onset of ventricular fibrillation means the death of the heart and of the patient but there are occasional cases one will never know how many who survive not only one but numerous attacks of that usually fatal arrhythmia. It can be distinguished from the ordinary Adams-Stokes syndrome due to a v block by electrocardiogram (or by the feel of the squirming, not pulsating, ventricles if the hand of the surgeon grasps the heart) and since the treatment of the two conditions is so completely different it is most important to obtain an electrical record during a syncopal attack if at all possible When ventricular fibrillation occurs as a complication during anesthesia or a surgical operation life may be saved by the quick application of a defibrillating electrical shock which should be available in as many operating rooms as possible The old time use of epinephrine by application to the heart directly inside or out, in emergency may be helpful in the usual cases of ventricular standstill but it is just the wrong therapy for ventricular fibrillation which indeed it may induce

If syncope due to ventricular or cardiac standstill or to ventricular fibrillation is prolonged it may lead to *convulsions.* In fact the Adams-Stokes syndrome is commonly defined as syncope and convulsions associated with marked slowing of the pulse. It is to be distinguished from epilepsy by the very marked pulse slowing in the former and the earlier occurrence or sequence of the convulsions in the latter

When faintness or syncope results from either tachycardia or cardiac (ventricular) standstill it is important to review the drugs being used. Excessive digitalis may induce a serious paroxysm of ventricular tachycardia and probably even ventricular fibrillation in susceptible sensitive or diseased hearts it may cause a v block or even atrial standstill although rarely the Adams-Stokes syndrome unless high grade partial block is already present. Quinidine sulphate can paralyze the s-a and a v pacemakers. Chloroform and related anesthetics can cause ventricular tachycardia and ventricular fibrillation Other drugs in poisonous doses can also produce arrhythmias, and therefore all therapy should be scrutinized when faintness and syncope appear to be caused by faulty heart action.

Related to faintness and syncope is a *state of shock* about which two important comments are here in order In the first place shock may be induced by several serious cardiovascular accidents. The most common of these is acute coronary thrombosis. Two other accidents responsible for shock in occasional cases are acute pulmonary edema due to left ventricular failure or to an abnormal tachycardia in mitral stenosis and acute usually massive pulmonary embolism Rare causes are dissection of the aortic wall and *rupture of the interventricular septum* or of heart valve or papillary muscle. The differential diagnosis of these various causes is as a rule fairly easy once one thinks of

the various possibilities and makes the necessary tests by history physical examination electrocardiogram and x ray

The other important comment about shock as a complication of cardiovascular disease is that it often masks other diagnostic symptoms such as pain palpitation and dyspnea and may therefore confuse the observer considerably As a rule however there is a short time, even if but a few minutes when the initial symptoms were present before shock occurred, and someone, a relative, a friend or a bystander may be able to tell the physician about this. I shall never forget two patients whom I saw in my early days one with intense breathlessness due to pulmonary edema and the other with the excruciating pain of massive myocardial infarction who in the course of an hour lost respectively their dyspnea and pain and quietly died in shock

In contrast to faintness, syncope and shock, *vertigo* is rarely related to heart disease. When intense it may be traced to the internal ear as Ménière s disease or to a cerebellar defect but it can be confused with a heart attack in a rare case not adequately appraised A very helpful clue in a severe attack of acute Ménière s disease is the desire of the patient to lie with his eyes closed Continuous buzzing in the ears should make one suspect an intracranial a v communication auscultation over the skull may reveal it. A cardiac or arterial murmur may be heard in a troublesome way in one ear or both especially if the ears are faulty also

Insomnia and extreme restlessness as chief complaints are uncommonly due to heart disease As a rule they are to be ascribed to hypersensitive nerves under strain or excessive stimulation from one cause or another but they can in rare cases of heart disease mask other symptoms

Very careful detailed observation may be needed to unravel clues. I have seen patients who were sleepless because of dyspnea (orthopnea or Cheyne-Stokes respiration) of which they did not specifically complain until asked they would sit up or walk the floor at night to get relief. Hypnotics would make them worse increasing Cheyne Stokes respiration for example or inducing nightmares or hallucinations One patient of mine was about to be sent to a psychopathic hospital because of these symptoms and reactions but fortunately was discovered to have incipient myocardial failure Digitalis and mercurial diuretics quickly cleared his insomnia and hallucinations and in a few days he was perfectly comfortable Digitalis itself unlike the hypnotics and narcotics does not cause hallucinations or psychoses although it has on occasion been blamed for disturbances of the mind due to the circulatory failure for which it is prescribed nor has it in my experience ever caused an allergic response although such is conceivably possible Annoying chest pain of coronary or aortic origin or due to neurocirculatory asthenia and bothersome palpitation can act in the same way with much benefit by treatment of the specific conditions back of the insomnia and restlessness.

Headaches and other intracranial circulatory disturbances sometimes lumped under the term *encephalopathy* are not characteristically found with heart disease per se or failure of either the myocardium or the coronary circulation Furthermore the vast majority of headaches are not associated with diseases either of the heart or of the blood vessels. Headaches are however common with hypertension and cerebral arteriosclerosis and in advanced cases may presage so-called little strokes consisting of transient paralyses, even a partial or complete hemiplegia lasting a few hours to a day or two and clearing wholly or in large

part, only to be followed by other attacks and often culminating in a massive stroke which may be terminal. One should mention in passing the sudden excruciating headache that often is followed by fatal bleeding in the case of rupture of a small (? congenital) aneurysm located in the Circle of Willis and called familiarly subarachnoid hemorrhage usually victims of this disease have no heart trouble but only the local cerebral vascular fault with or without hypertension

Aphasia word blindness and loss of memory may be transient or permanent results of cerebral vascular disease which fits into the category of either hypertensive or arteriosclerotic encephalopathy It is possible of course to have much the same effects of varying extent and of varying duration from cerebral embolism secondary as in the case of disorders of vision to intracardiac thrombosis.

Visual disturbances Sudden loss, partial or complete of the sight of one eye can result from embolism due to intracardiac thrombosis based on a variety of causes such as subacute bacterial endocarditis involving either aortic or mitral valve rheumatic heart disease and large myocardial infarcts or even cardiac dilatation of any type with failure especially if there is also atrial fibrillation. Or there may be thrombosis or hemorrhage with hypertension or arteriosclerosis.

An extensive and characteristic picture of retinal changes is found in malignant hypertension even with choked disc clearing surprisingly on occasion with treatment of various sorts sympathectomy hypotensive drug therapy and the rice diet. In fact the examination of the eyegrounds affords one of the best clues to the severity and prognosis of hypertension and to the efficacy of its treatment.

Flickering of the sight and colored vision may be due to digitalis intoxication and should always be looked for

if there are other suggestive symptoms such as anorexia nausea, or irritation of the colon or if unusually large doses of the drug are being administered

Cough and hoarseness have their origin rarely in diseases of the heart or great vessels but once in a while they are useful clues thereto. The former cough may be the result of pulmonary vascular congestion and especially pulmonary edema with the raising of frothy sometimes blood tinged sputum. Pressure from a very large heart, left atrium pericardial effusion, or aortic aneurysm can cause a bothersome chronic cough often brassy in character when an aneurysm is responsible In the vast majority of patients who complain of a cough however the heart and great vessels are not responsible even though they may be diseased In such cases the cough is due to an acute or chronic inflammation of bronchi lung tissue trachea, larynx, or upper air passages (with the dropping of secretions into the trachea) or to the irritation caused by tobacco In rare cases of myocardial failure with pulmonary vascular congestion cough is more bothersome than dyspnea and may be induced by recumbency as on first lying down at night

What has been said about cough applies also to hoarseness Very rare patients show unilateral laryngeal paralysis due to pressure on the recurrent laryngeal nerve from an aortic aneurysm or from a dilated pulmonary artery displaced upwards by a very large left atrium A good many years ago I happened to come across several patients with mitral stenosis and laryngeal paralysis but since then I have encountered very few more

Gastro-intestinal symptoms There are several such symptoms that can be blamed in rare cases on cardiovascular disease but clues are often not sought The first of these is *dysphagia* Only in the rarest cases is gross enlargement

For di[illegible] of [illegible] see the Appendix (page [illegible])

of the heart itself or a massive pericardial effusion big enough seriously to displace or to compress the esophagus which is a very adjustable more or less elastic tube. Much more responsible is displacement with compression by a very abnormal aorta as in the case of a large aneurysm or by a vascular ring due to a congenital anomaly in particular a right subclavian artery arising from the descending thoracic aorta The clues in analyzing this symptom are two first, the realization and remembrance of the possibility and second, x-ray studies with barium in the esophagus. It is important to establish the diagnosis since nowadays surgical therapy may be effective

Cardiospasm with or without the belching of gas is not a cardiac symptom but may simulate angina pectoris and may complicate heart disease of any type. It has been discussed in Chapter 5 to which the reader is referred. In brief it is the discomfort substernal in location produced by spasm of the esophagus and cardia of the stomach Its causes are myriad. One of the exciting factors in cardiospasm is angina pectoris. This has been so confusing in the past that on occasion esophageal spasm has been considered to be the reflex mechanism of the symptom of angina pectoris set off by myocardial anoxia

Anorexia is not a cardiac symptom but it may be an important early clue to digitalis or other drug intoxication. Of course advanced heart failure and the most acute stage of coronary thrombosis may be accompanied by anorexia

Nausea and vomiting may be initiated by a heart attack of any sort especially by acute coronary thrombosis but more commonly it is the medication rather than the disease that is responsible Morphine and related drugs and digitalis are common causes, and quinidine too may be responsible I have found it advisable to use as little opiate as possible in treating the pain of acute coronary thrombosis since the vomiting that can ensue may be more dis-

tressing than the pain itself and may at such a critical time actually jeopardize life. Finally it should be observed that there may occur simultaneously a heart attack such as coronary thrombosis and an abdominal disease in particular cholecystitis with gall stones, primarily responsible for the nausea and vomiting and for abdominal pain

Abdominal pain is rarely caused by cardiovascular disease The exceptions are 1) an epigastric location of the oppressive pain of coronary insufficiency or thrombosis with or without radiation upwards or to the arms 2) pain and tenderness over a liver engorged acutely by heart failure or pericardial effusion 3) arterial embolism to an abdominal branch of the aorta or to its bifurcation with obstruction of blood flow and 4) dissection of the aortic wall or pressure from or rupture of an abdominal aneurysm These conditions are sometimes very difficult indeed even impossible to diagnose but the presence of heart or aortic disease or of pericarditis should always cause a consideration of the possibility that one of these complications may explain an abdominal emergency not readily accounted for otherwise

Gaseous distention of the abdomen is a very common accompaniment of heart disease and is usually unrelated to it but it can be produced or aggravated when there is congestive heart failure or during the few days immediately following an acute coronary thrombosis as the result of the narcotic therapy the absolute rest and the abrupt change in diet

Irritability of the colon even with diarrhea may be caused by digitalis or quinidine intoxication and in some cases thereof is more prominent than nausea

Urinary symptoms are occasionally found in cardiac patients, usually coincidentally when there is prostatic obstruction with retention or when there is urinary tract

infection or nephritis. However such troubles are aggravated when there is congestive heart failure or acute coronary thrombosis, and it may be necessary to institute catheter drainage and antibiotic therapy for some days or weeks. Hematuria may follow renal infarction due to embolism from an intracardiac thrombus and is then usually relatively painless in contrast to the hematuria which accompanies renal colic caused by calculi. It should be added that bleeding from the kidneys as well as from the gastrointestinal and respiratory tracts may result from inadequate control of the prothrombin time when anticoagulants are freely used, as for coronary or other thrombosis or embolism. One other urinary symptom of interest is an increased frequency or amount caused by the reflex effect of paroxysms of tachycardia in some patients with such arrhythmia. It should also be remembered that a copious diuresis induced by treatment, in particular by mercurial diuretics, may be superimposed on an obstructing prostate to cause much distress unless it is quickly noted and relieved.

Sweating is a common symptom in heart disease as it is in many other conditions. As a "cold sweat" it often accompanies the state of shock in serious cases of acute coronary thrombosis or acute pulmonary edema. It occurs also in most patients with dissecting aortic aneurysms and in many with pulmonary embolism. Without shock it may accompany distressing paroxysms of tachycardia and it is common in neurocirculatory asthenia, thyrotoxicosis, and hypertensive crises. In unusual cases it may also accompany and even rarely presage attacks of coronary insufficiency and myocardial failure, for example at night without obvious pulmonary edema; in these last mentioned cases the upright position affords symptomatic relief.

Finally *weakness* is a symptom often complained of by

patients with heart disease but except in a few individuals with marked aortic stenosis limiting the cardiac output and so the blood supply to the body tissues it is not a cardiac symptom per se it is related otherwise to the circulatory system only in advanced cases exhausted by their disease Much more often it is but a symptom of nervous fatigue of neurocirculatory asthenia, of physical unfitness due to an invalid life, or to some specific disease such as anemia or an infection that may complicate heart disease However undue fatigue in older patients can result from faulty circulation caused in part at least by heart disease and myocardial insufficiency

CHAPTER 8

SIGNS

LESS important on the whole than symptoms as clues to cardiovascular diagnosis and treatment are signs identified by physical examination. Although this statement is true in the aggregate there are many exceptions a single sign may on occasion be the one clue to a correct diagnosis. Those signs requiring instruments for their detection such as the stethoscope for cardiovascular sounds and murmurs and the sphygmomanometer for blood pressure determination will be discussed in the next two chapters, to be followed by the still more special technics of roentgenology and electrocardiography

In the search for signs that may be important clues one should, of course, carry out a systematic physical examination such as is routinely advisable in all cases anyway One can quickly cover the body from head to toe and in this chapter I shall follow that sequence. It is of interest historically to note that Theophilus Bonetus in the two large volumes of his Sepulchretum (First Edition 1679) established this systematic plan of case teaching

What signs of cardiovascular value can one pick up upon *examination of the head?* It is sometimes said that *premature grayness* and *premature baldness* are common among the young patients with coronary heart disease the former as evidence of early aging and the latter as a sign of masculinity the male being many times more often affected in

youth by serious coronary atherosclerosis than the female. Although this may be true, there are so many exceptions to this statement from both points of view that one cannot *place much of any reliance on it.* Somewhat the same thing may possibly be said about relative lack of a beard as a feminine trait and therefore a point against coronary atherosclerosis but that too has not as yet been adequately studied.

The color of the hair and eyes has sometimes been mentioned as more or less characteristic of certain types of heart disease, but I have not found this to be of any value nor freckling of the skin nor pallor nor the brunette nor the presence or absence of acne. Observations pro and con all these findings are however still in the realm of conjecture since so far as I know there have been no adequate extensive systematic studies thereof.

There are however *five abnormalities of the skin* which can be helpful clues. The first of these is *pallor* which is often due to *anemia* and indicates the need of studying the blood since anemia itself not only is an important cause of heart murmurs which may simulate those of heart disease but also can result from subacute bacterial endocarditis or aggravate the effects of heart disease already present as in the case of coronary insufficiency. In one clinic my correspondent has written that the hematologist reports that more than 50 per cent of his untreated cases of pernicious anemia have been sent to him by the division of cardiology. Acute pallor is a common sign of severe coronary insufficiency.

Cyanosis when present is most obvious as a rule in the *mucous membranes of the mouth* but it may be apparent in the skin of the nose, ears, cheeks, fingers, and toes. The source is to be looked for in heart or lungs but it can be *physiologically produced in the skin by exposure to cold.* There may be some local circulatory fault to explain a local

cyanosis as from stasis due to a vascular defect, but the general causes are commonly pulmonary disease (emphysema extensive infection pulmonary arterial occlusion—embolism or thrombosis and an arteriovenous fistula) congenital defects with right to left shunts of large caliber and failure of either ventricle (the left causing pulmonary vascular engorgement and thus anoxemia the right causing peripheral circulatory stasis) Polycythemia increases cyanosis, anemia decreases it. Since in dark-skinned persons, especially in the black and brown races, the lips often are *highly pigmented it is important t examine the mucous* membranes of their mouths if cyanosis is suspected.

There is a skin coloration that may be confused with cyanosis although the differentiation is actually easy *Argyria* resulting from the chronic therapeutic use of *silver salts for nasal infections or gastric ills may* give a ghastly steel blue or gray color to the skin of the exposed parts of the body It is readily distinguished by the c lor itself which is not the purple blue of cyanosis by the fa t that it is more intense around the mouth and eyes by the obvious *absence of heart and lung disease and* by its m dification by exercise which impr ves the patient's c lor very much the gray then bec ming m re pink and more healthy in appearance while cyan sis due to heart r lung disease is usually increased by exercise

There are two more clues that may be derived from areful scrutiny of the skin. *Ja nd e* especially in the presence of congestive heart failure with engorgement f the liver suggests the possibility of the presence of a large hemorrhagic infarct f the lung or f rheumatic pneumonitis, while *pete hial h n orrhage* scattered over the body should make one look for subacute bacterial endocarditis if there is already valvular heart disease or a congenital defect of the heart n great vessels

The *eyes* sometimes yield valuable clues. *Exophthalmo*

may at once suggest thyrotoxicosis which can explain tachycardia, arrhythmia, and in advanced cases cardiac enlargement and failure. It may be interpolated here that "masked" hyperthyroidism really means that we are "masked" even though there may be little or no exophthalmos in the case under study. *Pupils* that are unequal or fail to react to light suggest central nervous system syphilis that may be accompanied by aortitis, aortic regurgitation or aneurysms. Characteristic *retinal changes* with arterial compression of veins, hemorrhages, retinitis, and even chocked discs point to the probability of a serious grade of hypertension which not infrequently has been more prominent in the past than in the present and which can explain left ventricular enlargement with or without failure. *Cataracts* and the *arcus senilis* which are often, of course, accompaniments and manifestations of the aging process are of little value as clues to coronary atherosclerosis; they may accompany heart disease but I have not found them helpful, many of my patients with serious coronary heart disease having neither, while many persons with either or both show no clinical evidence of heart disease. *Disturbances of vision* have already been discussed in Chapter 7.

Xanthelasma, which consists of small, slightly elevated, whitish spots of cholesterol deposition present in the skin of the upper part of the nose and around the eyes, is considered by some to be important confirmatory evidence of atherosclerosis of the coronary type.

The presence of *large scarred tonsils* was common with rheumatic heart disease many years ago but now one rarely sees them in anyone even with a normal heart because of their routine and often fully justified removal in early life. *Defective teeth* have little relationship to heart disease except that their removal may exhaust an older person with very limited myocardial or coronary reserve (if too

many teeth are taken out at one sitting) or may expose a young or even older person with congenital or rheumatic heart disease to subacute bacterial endocarditis through the invasion of the blood stream by the Streptococcus viridans via the raw gum surface unless enough penicillin is given prophylactically *Abnormalities of the ears* may sometimes reveal a clue. For example, a recent deafness or ringing in one ear or both may explain vertigo due to Menière's disease which on rare occasions, may be so severe that it can cause a profound prostration which can be mistaken for a "heart attack" (coronary thrombosis) a clue in differential diagnosis is that the patient with intense Ménière's disease prefers to lie flat *with his eyes closed* Sometimes extrasystoles or the postextrasystolic beats are felt in the ears when there is a faulty local circulation and in some cases a loud heart murmur especially one originating at the aortic valve as in aortic stenosis can be heard uncomfortably in one or both ears. Recently I have encountered a rare case of a roaring murmur in one ear (the left) due to arteriosclerotic narrowing of the left carotid artery in a diabetic patient of middle age the murmur (with thrill) over the carotid artery was of grade 5 intensity

The *neck* affords on occasion several valuable clues. A *goiter* with general enlargement or localized adenoma is often of course inert, but at times it can explain tachycardia arrhythmia and cardiac enlargement. One should look for any asymmetry of the thyroid gland in doubtful cases. A vigorous arterial (*carotid*) *pulse* is common with hypertension, a rigid sclerotic aorta and aortic regurgitation In cases of marked so-called free aortic regurgitation, whether syphilitic or rheumatic in origin the head may bob a little with each pulsation this has been called de Musset's sign Sometimes when the chest is short the innominate pulsation can be seen on the right, particularly

when the aorta and its branches are elongated and hyper tensive an aneurysm may then be simulated as a matter of fact, a real aortic aneurysm may extend to the neck also And thrills and murmurs may be transmitted to the neck from heart and aorta.

Jugular vein pulsation is always to be looked for with the subject seated it is often normally visible in the supine position When venous pulsation is visible in the neck in the upright posture it is an indication that something is wrong except in very rare instances of short chested, usually obese persons (generally women) in whom the superior vena caval or jugular pulse is just barely visible above the right clavicle Otherwise the commonest cause is congestive failure of the right ventricle with increased systemic venous pressure the greater the congestion the higher the pressure and the higher the venous pulse is seen in the neck extending even up to the right mastoid and styloid processes and into the cranium itself When the rhythm is normal one can usually easily identify three waves with each heart beat the *a* due to atrial contraction the *c* due to ventricular contraction and the *v* a stasis wave during ventricular systole ending with the beginning of diastole and the opening of the tricuspid valve

When the pressure in the jugular veins is very high they may not show much pulsation but are constantly distended as in the case of the so-called superior mediastinal syndrome due to marked compression or thrombosis of the superior vena cava as from an aortic aneurysm or to a high degree of chronic constrictive pericarditis or to excessive right ventricular failure The reverse finding of clearly visible and even marked ventricular systolic pulsation in the neck veins with little or no engorgement in diastole means free *tricuspid regurgitation with relatively little congestion* when this type of pulse is seen month after month with relatively little disability in a patient with chronic rheu

matic valvular disease it generally means that *tricuspid stenosis and regurgitation* are present as a fixed structural lesion accompanying mitral stenosis although in very rare cases it may be due to *irreversible dilatation of the tricuspid ring* with little or no valvular deformity per se Generally there is some slight increase in venous pressure even in diastole when one sees this chronic systolic jugular pulse and also the atria are usually fibrillating so that no *a* wave is visible only the *c* and *v* waves being seen

Finally two other comments should be added about the abnormal jugular pulse. The first is that in cases of chronic systolic pulsation the deep as well as the superficial veins are involved in fact usually much more so resulting in a forceful ventricular systolic elevation of the sternocleidomastoid muscle which may simulate carotid pulsation light pressure over the jugular bulb at the upper border of the right clavicle quickly obliterates it, however The second comment is that with much rise in venous pressure the *c* and *v* waves are not so distinctly separated as when the pressure is less and this results in one broad prolonged pulsation quite obviously longer in duration than the carotid pulse Mackenzie used to call this the ventricular type of jugular pulse but the congestive type would be a better designation since sometimes the rhythm is normal and the *a* waves are also seen.

Now we reach the *thorax* itself There are three deformities that can be helpful clues. The first appearing in childhood is that of a *bulging of the precordium* with actual bowing out of the ribs due to early enlargement of the heart congenitally of the right ventricle or of the whole heart in cases of severe rheumatic fever The second is the *protrusion of an aortic aneurysm* high under the sternum and costal cartilages on either side this was much more

For discussion of *Examination of the lungs and pleurae* see the Appendix (page 171)

often seen a generation ago than now following the impressive control of syphilis itself. The third is the *kyphosis* with deepening of the thorax that one sees in *pulmonary emphysema* which may be responsible for dyspnea or the kyphoscoliosis due to early deformity which in itself leads not infrequently to the cor pulmonale the kyphosis that occurs as a manifestation of extreme old age does not have any important significance

Besides deformities of the chest there are *pulsations* that can be helpful diagnostic signs. The *apical impulse* of a very large heart, for example a cor bovinum can be seen *and felt in the axilla in the sixth and seventh spaces, and* between that extreme and the normal there are many degrees of enlargement with outward or downward displacement of the apex beat. One must, of course, take into account cardiac displacement to either side by hydrothorax or pneumothorax. When the chest wall is thin one can feel and rarely even see the impulse produced on either side of the upper sternum by the slapping shut of the pulmonary valve on the left or of the aortic valve on the right when there is increased pressure in the pulmonary or in the systemic circulation with accentuation of the second sounds. *When the heart is very large the whole chest may vibrate with the heart beat* especially when the atria are *so large that they extend almost to the right chest wall.* There can then be a see-saw pulsation the ventricular walls retracting from contact with the left chest wall during systole and thrusting the atria out to the right, especially when the tricuspid and mitral values are incompetent as they usually are in the case of such large hearts with diastole there is a rebound in the other direction thus producing a back and forth major pulsation of the left and right chest walls.

Normally the right ventricle in its contraction leaves the

chest wall while the apex of the left ventricle comes up and forward to strike the chest wall When the right ventricle is very large it makes up so much of the thickened anterior portion of the heart that the chest wall anteriorly at the left of the sternum is thrust forward, while over the apex it retracts in systole simulating the *Broadbent sign* which consists of a systolic retraction of the left ribs and inter spaces due to pulling in of the wall by chronic pericardial adhesions. Careful study is needed to differentiate these *conditions, but it is much more important to diagnose* chronic constrictive pericarditis by other signs (engorge ment of neck veins and liver a paradoxical arterial pulse and abnormal *T* waves in the electrocardiogram with very limited pulsation and sometimes calcification of the x ray heart shadow in the absence of any other evidence of heart disease per se)

The importance of *palpable thrills* has been overempha sized One finds them over the precordium or larger arter ies in thorax or neck accompanying louder murmurs in particular the typical murmurs of aortic, pulmonary and mitral stenosis, and of a ventricular septal defect. Once in a while one may pick up a continuous thrill by palpa tion of various parts of the body (accompanying, of course a continuous murmur) This may be felt over a patent duc tus arteriosus, in the neck when there is a loud venous hum (see Chapter 9) over the aortic area after rupture of the aorta into the pulmonary artery and anywhere in the body (head neck trunk or extremities) over an arteriovenous fistula or aneurysm congenital or acquired

Heart disease is not a cause of *tenderness over the thorax* even when there are symptoms of pain or dyspnea Neuro circulatory asthenia is a common cause of precordial tender ness. Tender spots are sometimes found over pulmonary infarcts. And injuries and neuritis (especially herpes zoster)

are important causes of severe local tenderness.

Examination of the abdomen may yield some helpful clues and should never be omitted, especially in cases of uncertain diagnosis. Palpation of the *liver* may disclose three abnormalities *enlargement* tenderness and pulsation. Ballottement has been used helpfully in rapid determination of liver size and tenderness. It is important to distinguish between a big liver and one that is low in position many errors have been made in this respect when the edge of the liver is felt a few centimeters below the costal margin It is wise always by percussion or x ray examination to determine the position of the upper border of the liver when the lower edge is well below its usual normal position at the costal border or a bit lower down. If the liver is found definitely to be enlarged and intrinsic disease of the liver is unlikely one should try to decide if a weak enlarged heart is responsible Failure of the right ventricle and constrictive pericarditis, acute or chronic, may show themselves first or to a preponderant degree in the liver which acts like a sponge Jugular vein engorgement and pulsation usually quickly follow or indeed may coincide with the enlargement of the congested liver And finally the legs swell A sequel very late as a rule is the development of *congestive ascites* in heart failure or constrictive pericarditis.

Liver tenderness is common when the liver first begins to enlarge due to stretching of the capsule and *right upper quadrant pain on effort* is one of the earliest evidences of failure of the right ventricle In mitral stenosis for example After the liver enlargement has become chronic, the liver is as a rule no longer tender or painful Cirrhosis of the liver is rare in cardiac patients but chronic congestion that has lasted for a good many years, as in the case of chronic constrictive pericarditis may induce some "cardiac cirrhosis.

Pulsation of the liver of slight degree is common but not easily detected; only pronounced pulsation becomes obvious on palpation or by graphic record, as in cases of tricuspid regurgitation without much chronic congestion. The pulsation, of course, represents, as in the neck veins, the waves transmitted from the right atrium, but via the inferior instead of the superior vena cava.

Splenic enlargement is common in noncardiac patients but when present in heart disease it should always make one think of and look for two conditions: 1) subacute bacterial endocarditis and 2) infarction from embolism arising from intracardiac thrombosis of other nature. With fresh infarction the spleen may be tender but the chronically hard spleen is not. Only rarely does the cardiac spleen become very large. It is interesting that although at autopsy a case of congestive heart failure may show slight splenic congestion and enlargement, clinical evidence thereof is uncommon even though the congested liver may be very large.

It is always well to palpate the *kidneys* for in rare cases surgical kidney disease may explain hypertension which may be relieved by nephrectomy and of course an enlarged infected kidney may explain the reason for the poorer state of health of some cardiac patient as may also a distended bladder, the result of prostatic obstruction.

One other important clue on abdominal examination which may explain obscure pain is the discovery of an *aneurysm of the abdominal aorta*. Enlargement, increased pulsation and tenderness may be quite evident and since it has become possible surgically to replace by grafts abnormal sections of the aorta, as in the case of an aneurysm or coarctation, it is no longer of only academic interest to make a diagnosis of an aneurysm of the abdominal aorta.

The *extremities* can afford vital clues to cardiovascular diagnosis. There may be weakness of arm and leg on one

side with hyperactive reflexes confirming or pointing to an old cerebral vascular accident. The *knee jerks* should always be tested their complete absence suggests central nervous system and perhaps aortic syphilis their excessive reaction like the finding of fingernail biting in adults indicates a hypersensitive nervous state often found in neurocirculatory asthenia Whiteness and numbness on exposure of the hands to cold supports a diagnosis of Raynaud s disease

Cyanosis of fingers and toes is found under the same circumstances as that of the face and mucous membranes, in certain cases of congenital heart disease (most commonly *with the tetralogy of Fallot) and in patients with serious* pulmonary disease or heart disease with advanced failure of either ventricle or both *Clubbing of fingers and toes* is found in the majority of cases of the cyanotic type of congenital heart disease (most commonly the tetralogy of Fallot) and in subacute bacterial endocarditis without accompanying cyanosis *it may however be a family trait* in the absence of disease of heart or lungs. Splinter hemorrhages under the nails may be found with *petechial hemorrhages* usually elsewhere too in subacute bacterial endocarditis. *Xanthomata* in the tendons of young patients strongly support a suspected diagnosis of coronary heart disease and should be carefully looked for in doubtful cases.

Remember that *edema of the legs* is due to heart failure in the minority of cases there are many other causes, especially obesity varicose veins and phlebothrombosis.

Finally we come to the blood vessels themselves in arms and legs. The *veins* (and the arteries too) are much more likely to be abnormal in the legs than in the arms. There are two chief venous abnormalities 1) varicose veins which cause trouble locally only except when so much blood

settles in large plexuses that the person so affected easily feels faint with orthostatic hypotension and 2) acute thrombophlebitis in the calves threatening to lead to serious pulmonary embolism In the latter case one calf or both tend to be swollen and tender and are painful when the foot is flexed (Homan's sign)

The *arteries* are important for two reasons their condition and their pulsation. The pulse should always be felt for in the feet (in the posterior tibial and dorsalis pedis arteries). If the pulse is not felt there it is important in some cases to palpate the popliteal and femoral arteries. Absent or much diminished pulses in the legs may be due to local blocking by sclerosis, endarteritis, thrombosis, or embolism or to coarctation of the aorta When there is a pronounced difference between the pulsations in arms and legs or between the arms blood pressures in all four extremities should be taken for more definite information. Marked differences between the radial pulses may be due to congenital vascular anomalies to pressure on the subclavian or brachial arteries from tumor or aneurysm or to atherosclerotic changes in the vessels themselves. The arteries may be sclerotic (hard) tortuous or beaded and can often be clearly outlined on an x ray film I have found by observations of thousands of patients over many years that by and large persons with sclerosed peripheral arteries, particularly the radials have not much clinical evidence of coronary atherosclerosis in their early lives, while the great majority of my young and middle aged victims of coronary heart disease have soft radials there thus seems actually to be a reversed ratio

One of the two technics that comprised the total examination in ancient days was palpation of the pulse to note its character rhythm and rate the other technic was uroscopy Undoubtedly our medical ancestors a few hundred

years ago paid much more attention to the pulse than we do today but, of course not much was known to explain the variations then. The rhythm and the rate have already been discussed in Chapter 6 (*Palpitation*) and will be referred to again in Chapter 19 (Electrocardiography) The pressure will be discussed in Chapter 10 (Blood Pressure) The *shape of the pulse waves* will be the subject of some of the remaining sentences of this chapter A *small thready pulse* is found in shock, in moribund patients, and in some cases of excessive tachycardia A *full and bounding pulse* is found often after exercise in vasodilatation from any cause e g alcoholic drinking in cases with much generalized arteriosclerosis including rigid aorta and large arteries, with patency of the ductus arteriosus of high degree or with a large arteriovenous fistula and at its highest degree in free aortic regurgitation when it is then called the *water hammer or Corrigan pulse* The *hyperdicrotic pulse is found in some fevers and other causes of* marked vasodilatation The opposite finding of a *plateau* or *anacrotic pulse* is most commonly caused by aortic stenosis. The *paradoxical pulse* which when of marked degree tends to disappear on full inspiration favors a diagnosis of cardiac constriction acute or chronic, including a tamponade The most important of all abnormalities of the pulse is that called *alternans* in which with normal rhythm there is a regular alternation of blood pressure levels and thus of fulness most easily determined by sphygmomanometry but also sometimes palpable pulsus alternans is indicative of left ventricular weakness.

A high fever e g 101 F or 10 C, and a high leukocytosis, e g 25 000 or more suggest as a rule some other diagnosis than either acute myocardial infarction or rheumatic fever when such conditions are the cause they must be regarded as unusually serious with poorer prognosis.

There may be appended here best in this chapter other important clues derived from technical studies not already entered in the chapters on roentgenology and electrocardiography or referred to under other chapters as in the case of the significance of the pulmonary blood pressure in Chapter 10 One of these is that a normal (or indeed increased) circulation time (or warm hands and legs) with obvious congestion suggests one of the high output types of failure—anemia thyrotoxicosis, or beriberi Increased oxygen in the blood in the right atrium as determined by cardiac catheterization, indicates the probability of an atrial septal defect, in the right ventricle a ventricular septal defect, and in the pulmonary artery a patent ductus arteriosus.

CHAPTER 9

HEART SOUNDS AND MURMURS

This chapter is not intended to be a treatise on auscultation but rather a presentation of the more important clues to cardiovascular diagnosis and treatment that can be derived from listening over the heart and blood vessels At the outset it is to be recognized that serious heart disease may be present, especially that of coronary nature with nothing to be found wrong on physical examination including auscultation Also it is true that much experience is needed not only to hear and time variations from the normal heart sounds and to pick up certain murmurs, but also to interpret them correctly Some observers still recommend direct auscultation especially in cases with very faint diastolic murmurs due to aortic regurgitation And finally the hearing ability of different observers varies greatly some peoples ears cannot hear for example very low pitched mitral diastolic murmurs that are quite obvious to others.

Let us discuss the *heart sounds* first A *much accentuated first sound at the apex* especially in the presence of congestive heart failure always suggests the possibility of mitral stenosis even though no murmurs may be heard at first and even though it can be caused in a good many cases by a hyperactive heart in a person with a thin chest wall It is well in such patients to listen to the heart with

the subject lying in the left lateral position after some exer cise at which time an apical middiast lic murmur may be come audible.

Accentuation of the pulmonary second sound (P +) is common normally in young people after exercise but whenever there is suspicious evidence otherwise of increased pulmonary arterial pressure at rest this sign can often be confirmatory Such increased pulmonary artery pressure may occur with mitral stenosis left ventricular failure and the cor pulmonale In such cases the pulmonary second sound is not only accentuated but often doubled also In patients with long standing hypertension and accentuation of the aortic second sound an important clue to beginning left heart failure is to be derived from the increasing loud ness of the pulmonary second sound which may eventually become even more accentuated than the aortic second sound (P + + greater than A +).

Increase in intensity of the aortic second sound (A_2+) is of course, a long known sign of systemic arterial hyperten sion but a change in character of the sound is also of im portance and is not infrequently found in cases with loss of elasticity of the aortic wall due either to advanced sclerosis or to syphilis There is then *more of a slapping*, almost metallic quality to the sound reminding one some what of the sound of a water hammer

Decrease in intensity of the heart sounds in gen [illegible] accompanies five [illegible] nd [illegible] m unusual thickness of the [illegible] wall as in the case of gross obesity pulmonary emphys[illegible] with considerable lung tissue between heart and chest [illegible] large pericardial eff usions, feebleness of the heart [illegible] and vascular shock These can usually be easily [illegible] entiated sometimes they are combined. A dec[illegible] intensity of a single sound may also be importan[illegible] a diminished first sound at the apex without [illegible]

heart sounds otherwise may denote myocardial weakness. A much decreased or even absent aortic second sound is commonly found in cases of considerable aortic valve stenosis similarly pulmonary valve stenosis (congenital) often results in a much diminished pulmonary second sound.

A *third heart sound* may be heard normally at the apex in an occasional active young person in good health and as a frequent finding in the presence of mitral stenosis, but in a person with heart disease and enlargement due to any other cause a loud third sound at the apex indicates as a rule dilatation of the left ventricle on the verge of failure or actually showing congestion under such conditions, and when the heart rate is rather rapid it give rises to the familiar *diastolic gallop rhythm* one of the most important clues to left ventricular weakness. Similarly a loud third sound at the lower end of the sternum giving rise to a diastolic gallop there is very suggestive of right ventricular weakness and dilatation which should be looked for by other tests, especially x ray and electrocardiographic. Left ventricular failure mitral stenosis and chronic constrictive pericarditis are important causes of the much increased right ventricular third sound. In the case of mitral stenosis the diastolic third sound is usually followed by the so-called middiastolic rumble which may or may not be prolonged into a presystolic murmur

A third sound may also be heard in systole in rare cases who seem to be in good health and is frequently referred to then as a *midsystolic click* its mechanism is obscure but it has no clinical importance except that it may be misinterpreted by the physician as a serious sign rendering it then a source of apprehension on the part of the patient

When complete heart block is present careful auscultation at the left sternal border may reveal faint sounds due to the regular contraction of the atria during the long

ventricular diastolic pauses. This finding is of little importance except that it indicates that the atria are following a normal sinus rhythm and not fibrillating

Sounds over the arteries are of little importance By the time for example that the well known pistol shot is heard, other signs of free aortic regurgitation or extreme vasodilatation have established the diagnosis long before

Murmurs Systolic There are a few systolic murmurs that with rare exceptions are important clues and suggest in the first place or help to establish a cardiac diagnosis. Such a murmur is one that is maximal at the apex transmitted well to the left axilla and lung bases but not so well to the aortic valve area and of grade 2 or 3 or louder This means *mitral regurgitation* but neither the character nor the intensity of the murmur reveals the cause of the regurgitation that is, whether it is due to a structural deformity of the valve or to relative insufficiency resulting from left ventricular dilatation It is true however that the most intense murmurs of this sort (that is, of grade 5) usually attended by palpable thrills, do almost always indicate mitral valve disease

Another systolic murmur of greater diagnostic significance is a loud one of grade 4 or 5 usually with palpable systolic thrill maximal at the aortic area and widely transmitted especially to the vessels of the neck to the shoulders and spine and to the cardiac apex, but not well heard in the lung bases this murmur is diagnostic of *aortic stenosis* usually valvular but rarely infundibular in almost 100 per cent of the cases, the very infrequent exception being a patient with an aneurysm of the ascending aorta. It should be recognized however that even a lesser

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The grading of murmurs here is that ordinarily used grade 1 representing very slight intensity grade 2 slight grade 3 moderate grade 4 marked and grade 5 very marked.

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right ventricular contractions are forceful and the right atrium not so full of blood as to damp the retrograde flow

Systolic murmurs heard over the arteries without compression of the vessels, which in itself can cause murmurs, may now and then reveal clues to local abnormalities. Thus such a murmur sometimes with a thrill may be felt over an aneurysm anywhere in the body In rare cases arteriosclerotic narrowing of the lumen of an artery may produce a systolic murmur this is commonest in the case of the leg arteries but I have heard it rarely over a stenosed carotid or subclavian.

Diastolic murmurs are even more definitive than systolic murmurs in establishing specific diagnoses. Thus the localized middiastolic murmur at the apex with or without presystolic accentuation usually low pitched and rumbling (rarely blowing) in character and when of higher grades, 3 or more attended by a palpable thrill represents in the great majority of adults *mitral stenosis* of rheumatic origin There are however occasional youngsters with fresh or recent acute rheumatic fever and adults with free aortic regurgitation or rarely other cause for left ventricular enlargement who show slight grades of this same murmur caused by *relative mitral stenosis that is, a normal mitral* valve but a dilated left ventricular chamber when this murmur is found with free aortic regurgitation as in some cases of syphilitic *aortitis it has been called an Austin Flint murmur*

Rarely tricuspid stenosis gives rise to a middiastolic murmur localized at the lower end of the sternum Also I have heard on two or three occasions a right sided i.e right ventricular Austin Flint murmur

An early diastolic murmur heard at the aortic valve area but best long the left sternal border and not infrequently at the apex, and of any grade of intensity means *aortic valve regurgitation* this is due in the great majority of

cases to aortic valve deformity rheumatic or syphilitic, but in rare instances to merely a more or less temporary or variable stretching of the aortic valve ring caused by hyper tension or by a loss of elasticity of aorta secondary to arteriosclerosis or syphilis A test has been proposed for the diagnosis of relative aortic regurgitation a drop in blood pressure caused by the administration of nitroglycerine may abolish the slight diastolic murmur and restore a clear cut aortic second sound.

When a similar diastolic murmur is heard best in the pulmonary valve area and just below it, following as a rule an accentuated pulmonary second sound, it is caused by *pulmonary regurgitation* In contrast to aortic regurgita tion pulmonary regurgitation is rare and is usually due to relative valvular insufficiency from pulmonary hypertension *in a minority of cases it is due to pulmonary valve* deformity secondary to a congenital defect or to rheumatic involvement. Pulmonary hypertension sufficient to cause pulmonary regurgitation is due as a rule to mitral stenosis or to left ventricular failure rarely is it a part of the cor pulmonale or the result of an atrial septal defect when it is found with mitral stenosis it has been labeled the *Graham Steell murmur*

Vascular diastolic murmurs are of no importance As Duroziez sign such a murmur may be heard over a major artery on slight compression when there is free aortic regurgitation

Continuous murmurs The easiest clues of all on auscultation come from continuous murmurs. That over the pulmonary valve is characteristic of *congenital patency of the ductus arteriosus* it may be of any intensity from grade 1 to grade 5 and if quite loud is likely to be accompanied by a palpable thrill A rare exception to this statement is the murmur of the same type that develops suddenly due to rupture of an aortic aneurysm into the pulmonary

artery usually in a middle-aged man who does not long survive this accident If this kind of murmur abruptly appears *to the right of the sternum in the aortic valve area, it means rupture of an aortic aneurysm into the superior* vena cava with the development of all the high venous pressure signs of the superior mediastinal syndrome When continuous murmurs are heard over other areas of the body—*trunk extremities, neck or head they are due to arteriovenous fistulas* (aneurysms) which may be congenital or acquired traumatic or not, except for that heard not uncommonly down the thoracic spine due to *congenital coarctation of the aorta* Inasmuch as arteriovenous fistulas anywhere *in the body are an important cause of cardiac strain and enlargement it behooves* the doctor when dealing with heart disease of apparently unknown cause to search the body for an unsuspected arteriovenous fistula, since the surgical correction of such a fistula can restore the heart to normal or at least relieve it of its serious strain

There is one very important potential cause for error in diagnosis in dealing with continuous murmurs and that is the misinterpretation of the *venous hum in the neck* which is a normal physiological phenomenon in many young children and even in a rare adult It may be very loud and roaring in character and attended by a thrill It is best *heard over the right side of the neck maximally just over* the clavicle and may be attended by a *thrill It is due to* the rush of blood down the jugular vein into the superior vena cava and may even be heard over the latter just to the right and sometimes even over the center and to the left *of the* upper sternum, which circumstance is a reason for *its occasional erroneous interpretation as patency of the* ductus arteriosus or aortic valvular disease. It can be easily and quickly obliterated by slight pressure over the jugular vein above the point of the maximal murmur or by having the subject change from the sitting to the supine position.

CHAPTER 10

BLOOD PRESSURE

LIKE auscultation the determination of the blood pressure may yield valuable clues to diagnosis and to the explanation of various symptoms and signs Before discussing the clues I shall interpolate a few observations about blood pressure itself

For completeness the blood pressure includes not only that in the systemic arteries representing the pressure in the left ventricle and aorta but also various other pressures namely that in the systemic arterioles and capillaries, that in the systemic veins, the venae cavae and right atrium that in the pulmonary artery and its branches representing the systolic pressure in the right ventricle that in the pulmonary veins representing the pressure in the left atrium and finally that in the portal system Also some would include the pressure in the lymphatic system and thoracic duct. Although it would be of interest and sometimes of value to know all these pressures for practical purposes and in the absence of simple technics for their determination we are for routine use limited to the measurement of brachial and sometimes of femoral arterial pressure to a rough estimation of the venous pressure in the jugular veins, and on occasion to a study by cardiac catheter of the pressure in the great veins the right atrium the right ventricle the pulmonary artery and the pulmonary arterioles.

Brachial arterial pressure Fortunately this blood pressure which is the most important clinically to measure is also the easiest to determine The various sphygmomanometers in routine use today whether of spring type or mercury column, are simple to manipulate and accurate enough for clinical use although they should be calibrated once in a while perhaps once every year or two or if they do not record easily An accuracy within 3 mm of mercury is sufficient since that much blood pressure range and even up to 5 or 6 mm. is of no real clinical importance

It is always advisable at the time of the first examination to measure the blood pressure in both arms. Often there is a slight difference not actually between the arms, but due to the time factor The first arm examined whether right or left is likely to show slightly higher pressures by 5 or 10 mm. than does the second arm whether this can be ascribed to a pressor effect of the first compression of an arm by the blood pressure cuff or to a nervous reflex from the patient s mind, or to something else, I do not know However no matter whichever arm one examines it is very wise if there is any suggestion of hypertension to repeat the test several times, best of all at the end of the total examination as well as early in order to find what the range is. Moreover it gives one much more information to record all the actual readings rather than to average them as simply one systolic pressure and one diastolic pressure or to record only the lowest reading which may be the last one

Nobody s blood pressure is static and the range of blood pressure between that which is called *basal* obtained at absolute rest, and that which is *maximal* obtained under varying pressor strains should be known whether there is hypertension or not At one time I measured the blood pressure of healthy young men when they first awoke in

the morning but while still in bed and again later in the midst of a busy day's activities and I found not infrequently *a range of 25 or 30 mm of mercury in systolic* levels This variation is even more pronounced in so-called hyperreactors and in hypertensives and may even exceed 50 mm one hypertensive patient that I remember came into the hospital tired and nervous one evening with a systolic pressure of 260 mm after a good night's sleep his pressure was 160 mm

Rarely is it necessary to measure and record the *blood pressure in the legs* Usually it suffices to feel both feet to determine the presence or absence of the pulses therein *(dorsalis pedis and posterior tibial)* and their fulness. If no pulse is felt in the foot the popliteal and femoral pulses may be palpated and the femoral arterial pressure measured for later comparison when dealing with certain conditions which may later improve either as the result of therapy surgical or medical or spontaneously such conditions include congenital coarctation of the aorta dissection of the aortic wall (the so-called dissecting aneurysm) saccular aneurysms of the abdominal aorta, iliac, or femoral arteries arterial embolism or thrombosis blocking the blood *supply to the legs and thromboangiitis obliterans or arteri*osclerotic narrowing of the main arteries to the legs Much more frequently than is the case of the arms the legs show a difference in pressure because of the much greater tendency of all the conditions just cited to involve the circulation in the legs than that in the arms. It should be noted that the femoral arterial pressure is normally greater than the brachial especially with the subject in the sitting or standing position

Diastolic pressure Although it is customary to discuss the systolic blood pressure first I shall not do so because of the much greater importance clinically of the diastolic

pressure. The reason for this greater importance is that the latter pressure more nearly than the systolic pressure represents the mean pressure which is exerting a constant strain on heart and arteries the three most important of these vessels are the coronaries, the cerebral and the renal. Every millimeter rise in diastolic pressure is equivalent to several millimeters rise in systolic pressure so far as these strains are concerned, although it is also doubtless true that a very high pulse pressure without much of any elevation of diastolic pressure but a considerable increase of systolic pressure can have a harmful water hammer effect on the arteries without much strain on the heart.

A diastolic brachial pressure constantly above 120 mm of mercury and frequently reaching heights of 150 or 160 or more bodes ill for heart, brain or kidneys and demands more than the simple routine treatment that may suffice for the great bulk of hypertensive patients. The systolic pressure in these cases is often not correspondingly high It may even be less than 200 mm so that the pulse pressure is relatively small even as little as 40 or 50 mm In my own practice about 10 per cent of patients with high blood pressure belong to this group and are as a rule good candidates for radical therapy Those of this sort under 50 or 55 years of age are mostly males and if they have reasonably good renal function and are not in a state of serious congestive failure they have as a rule been much helped by lumbodorsal (thoracolumbar) sympathectomy Other cases of this type or those who are too old or too ill for the operation or refuse it, have been benefited by a very low sodium diet (such as the rice diet represents in maximal degree) or by some of the new hypotensive drugs.

I would add two other observations about the diastolic blood pressure. In my own experience I have found that its measurement during the increasing compression of the

arm by the cuff on the way up to the systolic reading gives a lower level, as a rule by a few millimeters, than is true on the way down after measuring the systolic pressure first, with artifacts excluded Perhaps it would be best to note both levels, such as has been of late the custom in recording the diastolic pressure in cases of marked aortic regurgitation or extreme peripheral vasodilatation where there is a waterhammer or Corrigan pulse. My second observation concerns this measurement of the diastolic pressure in those two conditions. When there is a very distinct rather abrupt diminution in the sound on auscultation over the artery below the cuff or in the movement of mercury column or sphygmomanometer needle that level which is probably the diastolic pressure should be noted as well as zero where the sound may be still audible On occasion however no such abrupt diminution is noted and, therefore one should record only the zero.

Systolic pressure A generation ago this was the only pressure level measured and is all that most laymen know about today It is important in talking to patients about their blood pressure, especially to those who overemphasize the hazard of every additional millimeter of systolic level to explain the unimportance of exact figures and the great variability of the pressure both normally and in particular abnormally An increase of 3 to 4 mm on occasion has worried a good many persons quite needlessly and it is now very important to combat much of the blood pressure neurosis that has become common due to the little knowledge that the public has acquired

There are several phenomena concerned with systolic pressure that may occasion erroneous conclusions or afford helpful clues. One of these is the *auscultatory gap* This is a finding dependent on one method of sphygmomanometry that by auscultation and not present in the other

methods which should be used in doubtful cases as controls On occasion particularly in hypertensive patients, there may be a phase of silence or near silence of 30 or 40 mm of mercury during the time of the recording of the true pulse pressure that is the interval between the systolic and diastolic levels. After the measurement of the actual systolic pressure the auscultatory sound fades or disappears some 20 or 30 mm lower down and reappears after a drop of another 30 or 40 mm. only a moderate degree of pressure above the diastolic level Thus a hypertensive patient may have a systolic pressure of 190 mm while the upper level of the gap may register 170 and the lower level 130 with diastolic pressure at 100 Unless the cuff pressure in such a case is raised above 170 or the oscillations of the mercury column or dial needle observed as in the oscillometric method of sphygmomanometry or the radial pulse felt as in the palpatory method, an error in the recording of the systolic pressure is easily made being noted as 130 for example. The auscultatory gap is commonest in cases of hypertension or of aortic stenosis.

Remembering that an auscultatory gap is possible and utilizing the corrections just noted it should be easy to avoid an error of this sort There are various unproved explanations of the auscultatory gap which seems to have no clinical importance provided it does not cause an error in blood pressure measurement. The best clues to its discovery are to raise the pressure in the cuff to a high level say of about 300 mm. and to watch the oscillations of the sphygmomanometer

Respiration especially when deep, causes a change in blood pressure When it is thoracic in type and full there is normally a slight lowering of systolic pressure during inspiration and a slight rise during expiration. When a patient s pulse fades or indeed in rare cases disappears al

together during inspiration we have a maximal degree of this change due to the marked drop in systolic pressure This has been called the *paradoxical* pulse and is a valuable clue on occasion to constriction of the heart either acutely as in tamponade by blood or pericardial effusion or chronically as in the case of fibrous or calcific constrictive pericarditis. In either instance it is a clue to the need of therapeutic relief of the heart by evacuation of the fluid causing the tamponade or by resection of the chronically constricting pericardium

One other important clue in cardiovascular diagnosis obtained by sphygmomanometry although when of extreme degree also discoverable by palpation of the radial pulse is *pulsus alternans* Certain precautions in its identification are necessary In the first place the heart rhythm should be regular or disturbed only occasionally by premature beats which reveal or increase the alternation in the first 6 or 8 postextrasystolic beats secondly the effect of respiration should be ruled out, if necessary by the patient's holding his breath and thirdly the alternation must extend beyond the first three beats following an extrasystole The pulsus alternans may last for minutes at a time or even for days or weeks. It varies from 2 or 3 mm up to 10 or 12 or more. It is one of the most important clues which we possess of weakness of the left ventricle appearing early at times even in the absence of all congestion and yet it has been surprisingly neglected by the medical profession despite the ease of its discovery Every case of chronic rather severe hypertension, of aortic valve disease (either stenosis or regurgitation) of more than slight degree or of myocardial infarction being followed should be examined for pulsus alternans at the time of the periodic checkups. Its discovery warrants the use of digitalis and reduction of strain these measures may delay or prevent

the development of congestive heart failure

A few final remarks may be made about the systolic blood pressure. It is much more variable than the diastolic pressure being elevated much more by exercise excitement, or toxic agents. Physiologically it may be slightly to moderately increased when the pulse rate is very slow that is, under 40 per minute as in complete a v block or when there is free aortic regurgitation. Also it may be increased more than the diastolic pressure in cases of congenital coarctation of the aorta. If its considerable increase paroxysmally or constantly is accompanied by a high pulse rate and a much increased metabolic rate one should consider the possibility of a pheochromocytoma in contrast to the ordinary essential hypertension

Pulse pressure The pulse pressure which is the difference in millimeters mercury between the systolic and diastolic pressures affords a number of helpful clues Two of them which involve periodic variations largely dependent on the systolic pressure levels have already been discussed namely the *pulsus paradoxus* and the *pulsus alterna*[illegible] and the physiological increase of pulse pressure due to marked bradycardia has been mentioned. Among other clues are the following a marked usually temporary great *n r ase in pulse pressure* may occur in hypertension when both systolic and diastolic levels are raised this is due to the fact that the systolic pressure at such times of stress or toxic effect is raised much more than the diastolic. For example blood pressure readings that are ordinarily in the neighborhood of ?00 mm systolic and 120 diastolic may temporarily rise during the cold pressor test to [illegible]0 systolic and 150 diastolic, the pulse pressure increasing from 80 to 1?0. Or the increase in pulse pressure may be due more to a lowering of the diastolic pressure than to a rise of the systolic as in the case of *free aortic regurgitat on an arteriovenous*

fistula a rigid aorta or *extreme vasodilatation* Thus there may be only a slight elevation of systolic pressure to 160 mm. but a low diastolic pressure of 30 giving a pulse pressure of 130 Sometimes hypertension may complicate aortic regurgitation giving a systolic reading of 180 to 200 with diastolic level of zero.

One of the most important conditions leading to a chronically increased pulse pressure is that called arteriosclerotic hypertension here the systolic pressure is high often 200 mm or more and the diastolic pressure normal or only slightly elevated, say 90 to 100 In this condition there is relatively little strain on the heart and kidneys and many patients survive with it for a good many years, although quite likely it is something of a hazard when the cerebral arteries are brittle

A *decrease of pulse pressure* is found in a number of conditions and requires other information to establish the differential diagnosis. Thus, when both systolic and diastolic pressures are low especially the systolic we may find that we are dealing with chronic adrenal insufficiency as in *Addison s disease* or with acute prostration or *shock* Blood pressure readings in such cases may be in the neighborhood of 80 mm systolic and 55 diastolic with feeble pulse and a pulse pressure of only 25 instead of the average normal of about 50 In cases of high grade *aortic stenosis* the pulse pressure is small with a relatively low systolic level thus the systolic pressure may be 105 mm and the diastolic level 80 but now and then there is an auscultatory gap in this condition and the systolic pressure may be higher than this with a correspondingly larger pulse pressure. An anacrotic character of the pulse with a slow rise and a plateau summit help much in the diagnosis. Even in *marked mitral stenosis the pulse pressure may be quite* small due to a low systolic level.

Pulmonary vascular pressure. As already stated in the chapter on auscultation the intensity of the pulmonary second sound gives a useful clue to the degree of the pulmonary arterial pressure but to obtain even approximate actual figures cardiac catheterization is necessary. The normal range of the pressures in the pulmonary artery and right ventricle are as follows: 15 to 35 (average 25) mm. for the pulmonary artery systolic pressure and about 10 mm. for the diastolic and 15 to 35 mm. for the right ventricular systolic pressure and zero millimeters for the diastolic. An *increase in the pulmonary arterial pressure* is found with 1) left ventricular failure, 2) mitral stenosis (or regurgitation) and 3) pulmonary vascular obstruction as by thrombosis, embolism, endarteritis, or extensive pulmonary disease and destruction. A *decrease in the pulmonary arterial pressure* is found with any cause for collapse or shock and specifically in the case of congenital pulmonary valve or infundibular stenosis. An *increase of right ventricular pressure* occurs under the same conditions as those listed for increase in pulmonary artery pressure and in addition when there is pulmonary valve or infundibular stenosis while a *decrease in right ventricular pressure* is found in shock and with right ventricular failure.

Venous pressure. It is rarely necessary to measure the pressure in the veins. Years ago there were crude devices such as a glass capsule sealed over the skin. Nowadays when accurate measurements are required they are best and most easily obtained by intravenous or intracardiac catheter plus a strain gauge.

Systemic venous pressure. A rough but usually adequate estimation of the systemic venous pressure can be made by simple inspection of the jugular veins with the subject sitting vertically in the upright position. This jugular pressure is an index of the degree of congestion of the systemic

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carditis and 3) *obstruction* of either *vena cava* or both by mediastinal tumors or inflammation, or of the *right atrium* by tumor or clot, or *local obstruction* or *compression* of some particular vein. One of the chief clues distinguishing cirrhosis of the liver with or without ascites from chronic constrictive pericarditis is the high systemic venous pressure in the latter as clearly demonstrated by the engorged neck veins. In some cases with considerable tricuspid regurgitation there is a full venous pulse with a considerable pulse pressure which may actually be measured; this is found in cases of tricuspid valve disease or marked right ventricular dilatation. It gives rise to the important clue of chronic deep systolic jugular pulsation indicative in the majority of cases of tricuspid stenosis (and regurgitation) complicating mitral stenosis.

Decrease in systemic venous pressure has no clinical significance although it doubtless occurs in cases of starvation and oligemia from any cause.

Pulmonary venous pressure is not measurable except at the time of operations on the heart when pressure in the left atrium may be determined as indicative of that in the large pulmonary veins. Such pressure, both left atrial and pulmonary venous, is increased in cases of left ventricular failure, mitral valve disease, and thrombotic or tumor obstruction of the left atrium.

Portal pressure. Inasmuch as this pressure cannot be measured clinically we need only to recognize its increase in cases of advanced portal cirrhosis of the liver and splenic vein thrombosis, which conditions give rise to esophageal varices and to splenomegaly. Surgical alleviation is possible in some cases by anastomotic operations. The normal range of portal blood pressure, it is generally agreed, is 15 to 20 cms. of water. Anything in excess of 20 is considered to be elevated.

CHAPTER II

THE X RAY SHADOW OF THE HEART AND GREAT VESSELS

Once in a while although actually in but a small minority of patients x ray study of the heart great vessels, and lungs affords the clue to the diagnosis. Often it is simply confirmatory of the diagnosis already made clinically *On occasion it is negative failing to confirm a* diagnosis which had been made with the hope of x ray support, and as the result of which need of further study may be indicated For these various reasons I have found fluoroscopy essential in the routine examination of my patients even though it rarely affords the only clue to the correct diagnosis. I have found it helpful to observe the heart and aorta lung hiluses and diaphragm in action and to make a simple orthodiagraphic measurement of the transverse diameter of the heart shadow for future comparison under the same conditions so far as the examination itself is concerned. Of course such measurements are inaccurate up to about half a centimeter but it is helpful to have serial records of the transverse diameter as well as general descriptions of the fluoroscopic picture Best of all of course is the combination of teleroentgenogram ("7 foot film") or orthodiagram and fluoroscopy made serially at

yearly or more frequent intervals, although one can get along usually with the one technic or the other

As to clues the *determination of the heart size* is the most important. Not infrequently it is difficult or impossible *to find out how large the heart is on physical examination* because of obesity or pulmonary emphysema or cardiac displacement by one cause or another In such cases the x ray tube is invaluable A small or normal heart shadow helps very much to rule out heart disease as a cause of dyspnea or edema except in rare cases of mitral stenosis with paroxysmal dyspnea and not much enlarged left atrium and of chronic constrictive pericarditis with big liver and engorged neck veins. In such rare cases the clues are found elsewhere, by electrocardiogram and physical examination When the heart shadow is enlarged it is less helpful as a clue for then heart disease may or may not be the cause of dyspnea or edema that is other conditions such as pulmonary disease may complicate the heart disease and be entirely responsible for the dyspnea or leg vein thrombosis may be the complication to cause the edema

Enlargement of a particular part of the heart shadow may afford the clue Although it is not easy in many cases to distinguish between *left ventricular and right ventricular enlargement* or to determine the relative responsibilities of the two ventricles for an enlargement that is caused by both careful study *especially during the rotation of the* thorax under the fluoroscope can sometimes afford adequate information Left entricular enlargement increases the shadow posteriorly and downward as well as to the left (Fig. 1) while right ventricular enlargement increases the shadow anteriorly to hug the anterior chest wall as it were as well as horizontally to the left (Figs. 2 A and B) The shape of the chest and the height of the diaphragm always have to be taken into consideration A broad but

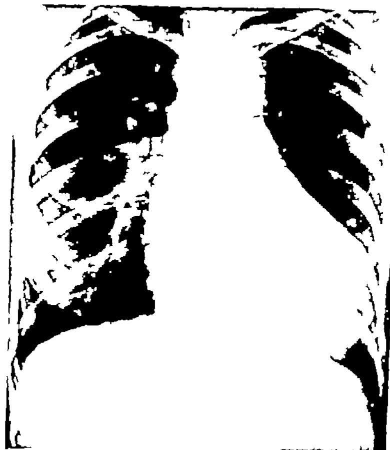


FIG. 1 M rked left entricular enlargement n oung woman th free aortic egurgita ion due to a congenitally biscuspid aortic al further damaged by subacute bacterial endocarditis. The slightly enlarged left atrium is sible through th heart shadow

short chested person with high diaphragm may seem t have a large heart when it is simply of full size and horizon tal in position while a tall person with long chest and low diaphragm may seem to have a normal sized heart because it is so vertical when actually it may be somewhat enlarged.

Incidentally it is in general better to speak of ventricular enlargement than of hypertrophy or dilatation alone inasmuch as in the great majority of cases with more than very slight enlargement both hypertrophy and dilatation are responsible.

Enlargement of the left atrium particularly in patients with mitral stenosis is a very helpful finding on x ray examination not simply to confirm the diagnosis but especially to ascertain the size of the left atrium which it is impossible to do by physical examination alone largely because that heart chamber is at the back of the heart and percussed only with difficulty (Figs 3A and B) On rare occasions roentgenology may reveal the left atrial enlargement of a mitral stenosis which is missed or indeed diagnosed only with the greatest difficulty on physical examination.

Enlargement of the right atrium of considerable degree is uncommon but it can be diagnosed by the x ray It is found in patients with tricuspid regurgitation or stenosis complicating mitral stenosis and atrial fibrillation and in cases with a large atrial septal defect.

The hugest hearts in volume are of rheumatic type with big right ventricle and very large left and right atria without much enlargement of the left ventricle (Fig 1). The shadow of such a heart sometimes almost fills the chest the borders closely approaching both walls. The *cor bovinum* (Fig 1) of aortic regurgitation is not so large in volume though twice as heavy its shadow bulges far downward and out toward the left axilla but not far into the right side of the chest.

A localized bulge of the left ventricular shadow (cardiac aneurysm) is sometimes seen in cases with large anterior or lateral *myocardial infarcts* (Fig 5) This may even include the apex when it is very large and so located Although the



Fig. .A. Pronounced right ventricula enlargement seen i .5 y old woman who ha alvular pulmonary stenosis nd patent forame ovale. Th characteristic post-stenotic dilatation of the p lmonary rtery and decreased vascularity f the lungs re present. The later l view show the right ventricle which has enlarged anteriorly nd is pressing against the sternum.

bulge itself is usually diagnostic its pulsation is a further clue when the rest of the ventricular heart shadow con tracts in systole the scarred region of the bulge dilates, thus



Fig. 21b. Lateral view

causing a *paradoxical pulsation* which can often be observed fluoroscopically If the scar is not so large as to cause an actual aneurysm it not infrequently seems to stand still in systole changing the contour of the so-called kymogram which records the movement of the heart border either on the film itself by the use of a grid or electrically by the use of a photoelectric cell it is difficult for any but experienced roentgenologists to detect a moderately sized or relatively

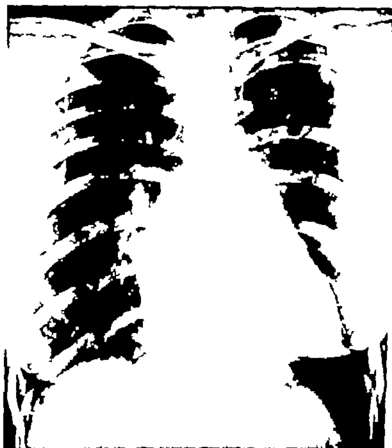


FIG. 3A. An enlarged left atrium seen projecting just beyond the right border of the heart in the frontal view and displacing the esophagus posteriorly in the lateral view. Enlargement of the pulmonary artery and of the right ventricle are also present. The patient had "pure" mitral stenosis which was relieved by mitral surgery. Barium is seen in the esophagus.

small scar of a myocardial infarct fluoroscopically.

An error *in interpretation of the x-ray film of the thorax* which used to be made frequently but which now is rather



FIG. 3B. Lateral iew

uncommon is to include in the heart shadow measurements a considerable *deposit of fat* along the left border and sometimes also along the right border This finding is most common in the case of a person who usually has a good deal of fat located elsewhere over the body and whose heart is often transversely or horizontally placed over a high diaphragm which position in itself often makes the



Fig. 4 Marked cardiac enlargement in a patient with mitral stenosis and regurgitation, tricuspid regurgitation and congestive failure. The left main bronchus is elevated by the enlarged left atrium.

heart shadow appear to be somewhat enlarged. The largest deposit of fat is usually at or near the apex where the heart shadow merges with that of the diaphragm (Fig 0). A *triangle of fat* several centimeters in transverse diameter may be seen at this pericardial diaphragmatic angle and be wrongly measured as a part of the heart shadow resulting



FIG. 5 A entricula neurysm projecting from the terior surfa of the heart nd best seen in the right terior oblique iew. Calcifica tion in the djacent myocardium or in mural thromb us is present. The patient had survi ed three myocardial infarctions

in an erroneous diagnosis of cardiac enlargement. The realization of the possibility of this finding in an obese person should put one on guard and a careful scrutiny of the film where a triangle of shadow less dense than the heart shadow may be seen to descend to the diaphragm



FIG. 6 A tri ngular fat pad at the cardiac pex n an obese patient This can giv n erroneou impression of cardiac enlargement The urved tour of the cardiac pex is seen to lie within the adipose tissue

diagonally towards the axilla should be enough to avoid an error in interpretation Best of all is to observe the heart and fat shadows fluoroscopically where the difference in density is more obvious and where the fat shadow can be displaced from the heart shadow during deep inspiration

Pericardial disease is diagnosable by x ray examination in a minority of cases but fortunately in the majority of patients who are seriously affected by it Acute pericarditis of virus origin, for example, or rheumatic in type causing pain and fever or a friction rub cannot usually be diagnosed by fluoroscopy or film If however a considerable *effusion* develops, that is of more than 250 cc serial records may reveal it. In the case of acute or subacute tuberculosis of the pericardium the heart shadow may slowly enlarge to enormous size with relatively little in the way of symptoms. Early evidence of tamponade occurs in patients who have a more rapid accumulation of fluid. Although large pericardial effusions have been credited with giving characteristic changes in shape of the heart shadow ("water bottle" for example) it is by no means easy to rely on such changes and the heart shadow may simply enlarge in general due to the superimposed pericardial fluid (Figs. 7A and B) As a matter of fact, not infrequently there is a combination of myocardial dilatation and pericardial effusion, as in some cases of acute rheumatic fever and then the differentiation by x ray becomes very difficult if not impossible. In such cases physical signs are more valuable pericardial friction rub paradoxical pulse liver enlargement, and fulness of the neck veins. A clue that is sometimes helpful fluoroscopically is a decrease of pulsation of the heart shadow in the case of tight pericardial effusions but on occasion a weak dilated heart may also fail to show much of any pulsation even if there is no increase of pericardial fluid.

Chronic adhesive pericarditis as in a case with rheumatic heart disease is usually not evident by x ray examination. When however symptoms and signs are caused by *chronic constrictive pericarditis* then it may be possible to confirm the diagnosis by the x ray By the time that roent



Fig 7 A. A large pericardial eff sion shown before nd fter pericardial tap with introduction f ir

genology affords the evidence of serious pericardial disease it should be possible to have already made the diagnosis clinically but once in a while the discovery of *pericardial calcification* (Figs. 8A and B) does lead one to look f r minor signs of constriction of the heart that might have been previously overlooked It should however be observed that calcification of the pericardium may be evi

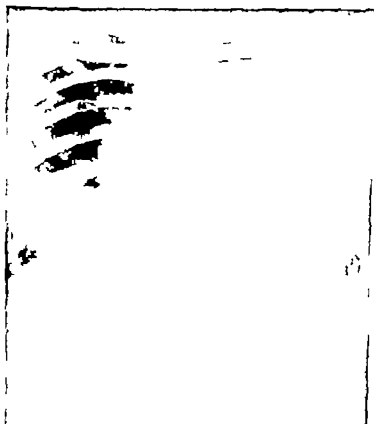


Fig 7B

dence of an old usually healed tuberculous lesion that in a few cases never reached a degree sufficient to cause constriction. Finally it is true that chronic constrictive pericarditis with or without calcification may so bind the heart by a shoe-leather like envelope that little or no pulsation of the heart shadow is visible fluoroscopically when a relatively small heart shadow in contrast to a much enlarged one is noted to pulsate little or not at all in the



Fig. 8A. Constrictive pericarditis in 50 year old man. Moderat cardiac enlargement nd rigl t pleural ff sion are present. \ I often the case calcification of the pericardium is more strikingly seen i the lateral iew Bari m is seen in the esophagus.

presence of signs of systemic venous congestion we are thereby afforded a very helpful clue. Of course in a pa tient with such congestion a small size of heart shadow is in itself at once suggestive of chronic constrictive pericarditis whether it pulsates or not in such cases abnormal *T* waves



FIG. 8B

in the electrocardiogram afford further evidence.

Now as to the shadows of the *great vessels*. Like the left atrium the *aorta* is largely out of reach of routine physical examination except when an aneurysm thereof presents itself in the anterior chest wall. Even most *aortic aneurysms* in the thorax are, however evident only by x ray examination. This is one of the reasons why I am never con-

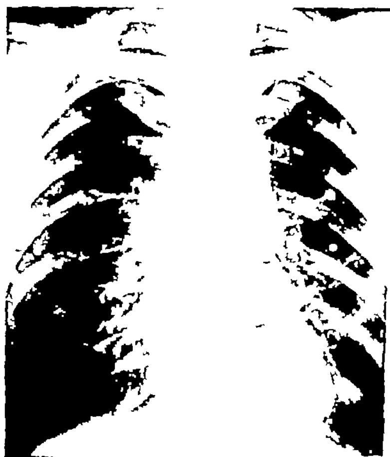


FIG. 10. Widening of the aortic arch and descending aorta due to dissecting aneurysm of the aorta in a hypertensive patient. A. *Before dissection.* B. The second film was taken 10 months after the first film and shortly following an episode of pain clinically consistent with aortic dissection.

without any coronary insufficiency (because of the development of an adequate collateral coronary circulation) and inasmuch as most patients with angina pectoris or coronary thrombosis do not have enough coronary calcification to be



FIG. 10B

visible by x ray this sign is not an important clue.

Dissection of the aortic wall (dissecting aneurysm) may be evident in rare cases roentgenologically by comparison of serial films, as noted above (see Figs. 10A and B) and in more or less unique cases a double barrelled aorta with calcified inner tube can be found. In obscure cases such should be looked for



FIG. 11 Widening and calcificatio I the ascendi g aorta du t syphilitic aortitis. This is best seen in the l ft nterior oblique view

There are two congenital aortic abnormalities that may be found by x ray examination The first is the *right sided aorta* which may be entirely unimportant clinically On occasion, however it may be associated with other congeni tal defects to produce trouble, as in the case of an aberrant *right subclavian artery arising from the descending aorta*

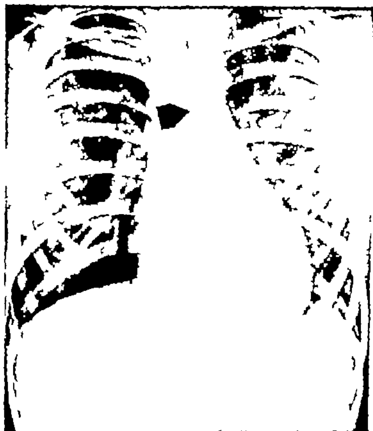


FIG. 12A. Coarctation of the aorta in a nine yea old boy ho a yet shows only slight notching of the ribs. The left entricle is a little large. The rea of oarctation ndic ted by the arrow is outlined against the bar m filled esophagus. Below this point there is slight post tenotic d l tation

and responsible for a constricting arterial ring around trachea and esophagus since surgical relief is possible in such cases not only should a right aortic arch be noted as suspicious evidence but also the contour of a barium



FIG. 12B. The second film illustrates coarctation of the aorta in older person, showing well marked notching of the ribs.

column in the esophagus (barium swallow) should be studied for an area of constriction

The other aortic anomaly is *congenital coarctation* which is a not uncommon cause of hypertension in children and young adults. There may be evident flattening or narrowing of the aortic knob or of the aortic shadow just below the knob in the x ray film (Fig 12A) and there may

be notching of the ribs in older children and adults due to erosion by the collaterally enlarged intercostal arteries (Fig 12B) often both of these may be noted Years ago the roentgenologist on occasion discovered coarctation of the aorta upon the finding of rib notching and called the attention of the internist or pediatrician to this diagnostic explanation of otherwise unexplained hypertension, but the alert practitioner of today should go to the x rays simply for confirmation of his clinical diagnosis. Nevertheless we do have useful clues in the x ray picture (Figs. 12A and B) The surgeon who is to correct a congenitally narrowed aorta is today naturally interested in its exact location and extent, and for this purpose on occasion a contrast dye aortogram is desired but since the procedure itself is not without some hazard it should probably be reserved for the more obscure cases

The *pulmonary artery* and its branches form an important part of the great vessel shadow above the heart itself and although great enlargement of the pulmonary artery can because of its anterior position in the thorax, become evident by percussion at the left of the upper sternum x ray study is necessary to establish a clear idea of its size and shape There are four chief causes of chronic and well marked enlargement of the so called pulmonary ar which includes the main pulmonary artery and the base of the left main branch the right branch being largely hidden by the aorta which overlies it These four causes are 1) pulmonary hypertension secondary to mitral stenosis 2) pulmonary hypertension secondary to pulmonary thrombosis or endarteritis obliterans and causing the enlargement of the right ventricle characteristic of the chronic cor pulmonale 3) a large left to right shunt of blood to overload the pulmonary circulation as in the case of a congenital atrial septal defect (Fig 13) or a patent ductus arteriosus

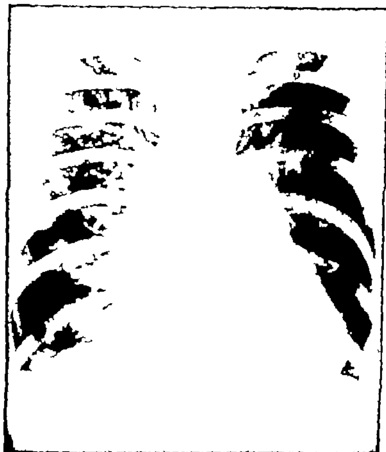


Fig. 13 Atrial septal defect showing marked enlargement of the main pulmonary rtery and its branches

(Fig. 14) and 4) pulmonary valve stenosis, congenital in origin with pulmonary artery dilatation just beyond the stenosis, even though the pulmonary blood pressure itself is low Pulmonary hypertension secondary to left ventricular failure can to be sure, cause an enlargement of the pulmonary artery also but this is usually neither very



FIG. 14 Patent ductus rteriosus l 10 yea ld girl with moderat enlargement of the mai pulmonary rtery nd increased scularity in the lungs

marked nor chronic because of the often rather rapidly ensuing failure of the right ventricle The condition wl ich produces the most marked enlargement of the pulmonary artery often with aneurysmal dilatation of its branches and which results in very wide lung hilus shadows is a large congenital atrial septal defect.

Enlarged lung hilus shadows are composed for the most



FIG. 15 A. A large dermoid cyst of the mediastinum illustrating its growth a 12 year period (from A to B). It was finally excised.

part of both dilated pulmonary arteries and dilated pulmonary veins when mitral stenosis and left ventricular failure are responsible In the other cases, the pulmonary arteries are the vessels involved Although actually the pulmonary arteries are located higher that is cephalad, in the mediastinum, there is not much distance between the levels of arteries and veins it is not always easy to differ



FIG. 15B.

entiate them fluoroscopically There is one condition which can be diagnosed or confirmed by fluoroscopy and not on an x ray film and that is pulmonary valve regurgitation which when of fair degree causes a considerable pulsation of the pulmonary artery and of the hilu shadows this igorous pulsation has been called the *hil r dance* when present it is a very helpful clue but it varies a good



FIG. 16 A. Severe pulmonary edema with dilatation f the perior ena ca and of the azygos ein in a young woman with mitral stenosis, who is now asymptomatic following mitral lve surgery In the second film, taken after clearing of the acute pulmonary edema an enlarged left trium is isible through the right portion of the heart shadow

deal in cases where there may be only transient relative pulmonary regurgitation that valve defect is responsible for a Graham Steell murmur which can similarly come and go (see Chapter 9)



FIG. 16B.

Mediastinal tumors may on occasion be differentiated from cardiovascular disease only with great difficulty by symptom sign or even by x ray study The greatest difficulty is in deciding whether a mass is a tumor (Figs. 15A and B) or an aneurysm since the former may transmit a contiguous pulsation and an aneurysmal sac may be thrombosed. With rare exceptions, however careful analysis of

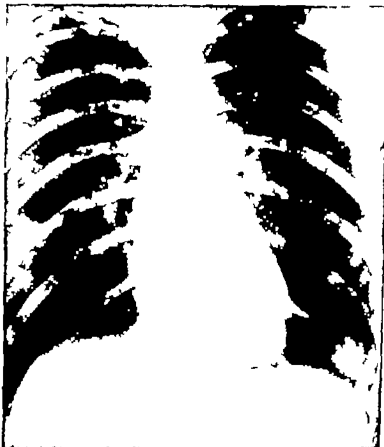


FIG. 17 A pulmonary arterio- enous fistula occurring i) ng woman with polycythemia and cyanosis The mass is isible t the left costophrenic angle A clue to its nature is given by the dilated essels running into the mass from the hilum of the left lung.

the patient from all points of view establishes the correct diagnosis one may need to use all possible clues.

It is important also not to confuse spinal scoliosis with enlargement or displacement of heart or aorta which is sometimes done when there is too hasty inspection of the

shadows in the thorax in the a p view either fluoroscopi cally or on the x ray film A correction is easily made by careful study of this view and by rotating tl e subject under *the fluoroscope or by taking films in the obli que positions.*

Two final comments should be made abc ut the pulmon ary circulation as observed by x ray *Pulmonary edema* when of considerable degree can show itself clearly as a diffuse mottling of the lung fields either generally or locally and the x-ray picture may demonstrate this easily (Fig. 16A) when physical examination of the lungs may reveal little Such x ray evidence of pulmonary edema like the edema itself can quickly come and go. The other point concerns the opposite picture namely that of unusually clear and bright lung fields due to a decrease of circulating blood in the lungs this may be found in a lung or a part of a lung distal to a point of pulmonary thrombosis and it is also seen in some cases of pulmonary stenosis where a less copious bronchial arterial supply to the lungs largely replaces the pulmonary arterial circulation

Finally the *great systemic veins* may be engorged in cases of failure of the right ventricle or of constrictive peri cardius, acute or chronic. The inferior ena ca a is not ordinarily visible unless it contains an injected radiopaque substance (diodrast) but the superior ena cava and the azygos veins can sometimes be identified when they are engorged (Figs 16 A and B). In rare cases there is a congenital persistence of a left superior vena cava draining into the coronary sinus and in doul tful cases this can be diagnosed by angiocardiography consisting of the injection of diodrast int the left arm vein.

In rare cases of pulmonary arterio-venous fistula it is possible to see the mass involved in the fistula in the x ray film (Fig 17).

For the more complicated technics of roentgenology

which may add further clues to diagnosis in rare cases the reader is referred to textbooks and works of reference in cardiology and roentgenology Such technics include angiocardiography cardiac catheterization electrokymography and tomography Examples of further clues are the visible passage of the cardiac catheter through an atrial septal defect from right atrium to left or into the aorta from the right ventricle in a case of the tetralogy of Fallot, and the quick demonstration of a thrombosed or compressed superior vena cava by diodrast injected into an arm vein.

CHAPTER 12

ELECTROCARDIO GRAPHIC CLUES

Much help in cardiovascular diagnosis comes from the electrocardiogram in the way that it is taken and interpreted today and yet when clinical electrocardiography was introduced some 40 years ago it had relatively little value being used simply to explain disturbances of cardiac rhythm. To be sure it continues to be very helpful in this respect but it has grown to be tenfold more useful in other ways. It is to present certain clues concerning all aspects of electrocardiography that this chapter has been prepared For a detailed description of electrocardiographic technics, basic principles, and interpretation the reader is referred to the special volumes on the subject.

In Chapters 6 on Palpitation and 13 on Therapy a discussion will be found concerning the symptoms and treatment of various *disorders of rhythm* all of which can be quickly and best identified by the electrocardiogram and which include *premature beats (atrial and ventricular) paroxysmal tachycardias atrial fibrillation* and *flutter* and *heart block* Despite the fact that it may seem quite evident clinically that an arrhythmia consists simply of premature beats it is often useful to obtain an electrocardiogram to rule out other abnormalities and thus to be able fully to

reassure the patient also to have at hand a record for future comparison and infrequently to uncover some other abnormality such as bundle branch block the presence of which although harmless in itself should be known

It is more important still to obtain an electrocardiogram during a paroxysm of tachycardia if it is frequent, prolonged or very troublesome in order to identify the type and also for future comparison Paroxysmal tachycardia of ventricular origin is generally much more serious than that of atrial origin and may be a manifestation of coronary heart disease or of digitalis intoxication. Sometimes a paroxysm consists of atrial fibrillation or atrial flutter which although on occasion occuring as a relatively simple disorder of function in a person without heart disease is much more likely to appear as a complication of heart disease than as a happening in the community at large Also the treatment of paroxysms long or short of atrial fibrillation and of atrial flutter is usually different from that of simple paroxysmal tachycardia and one can often follow the effect of treatment best by serial electrocardiograms. One example of the difference is that of the effect of carotid sinus pressure which should always be tested in a case with a paroxysm of atrial tachycardia since it is successful in stopping an attack at once in 5 to 10 per cent of patients while it is wholly ineffective in atrial fibrillation and may simply slow the ventricular rate temporarily in atrial flutter by increasing the grade of block from the usual 2 to 1 to 3 or 4 or more to 1 Another example is the greater effectiveness of digitalis therapy in atrial flutter than in paroxysmal tachycardia.

Before leaving the subject of atrial flutter I would like to cite the case of a patient whom I have recently seen with that arrhythmia. He exemplifies a number of features of that disorder which, although possible of interpretation by

studying the heart rate on physical exai nation, are inf n itely more easily and quickly analyzed I v electroca l 4 raphy He is a man aged 75 years wid a l l d grees of I pertension and arteriosclerosis but subje t to par ysms of atrial tachycardia every few weeks over the pa t several years During the paroxysms which have ften lasted a few hours each time he has been very cnscic us of the fast heart rate which has been recorded at 900 per minute or a little less With the faster rates he has noted a bilateral wristache. A fortnight ago he was found on rou tine ex amination to have a regular heart rate of 160 of wl ich l e himself was not aware. An electrocardiogram was taken which showed atrial flutter for the first time with an atrial rate of 320 and 2 to 1 a v block giving a ventricular rate of 160 a common finding in flutter Digitalis was begun in moderate dosage slowly to digitalize in addition t the quinidine rations which he had been taking three times a day for a year or more Two days later his ventricular rate was regular at 140 due to the slowing of the atrial rate to 280 by quinidine, the 2 to 1 block continuing. After another two days the heart rate had become regularly irregular with earlier beats every third or fourth beat clinically resembling premature beats but at the unusually rapid rate for the occurrence of extrasystoles of 108 to 110 An electrocardiogram quickly solved the mystery whicl could with more difficulty and much more clinical experi ence be solved without the help of the electrocardiograph The atrial flutter was still present but with much more a v block cycles of 4 to 1 block predominating but with occa sional more or less regularly recurrent 2 to 1 block (Fig 18) Quinidine was now stopped but the digitalis was continued and in another two days the ventricular rate was perfectly regular at 75 and the patient felt well An electrocardiogram showed an increase in the atrial rate to 300 (follow

ing the omission of the quinidine) but a steady 4 to 1 block due to the digitalis giving the apparently normal heart rate A continued use of digitalis should maintain a satisfactory ventricular rate whether flutter continues or changes to fibrillation or of course there may be a return to normal rhythm.

Finally so far as arrhythmias are concerned bradycardia of high degree, that is under 40 per minute, should always be studied electrocardiographically Although rare cases

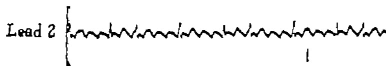


Fig. 18 Electrocardiogram showing trial flutter with trial rate of 320 nd ventricular trigeminal rhythm at rate f 96 due to alternating 4 to 1 4 to 1 and 2 to 1 block. A.F male ged 75.

especially highly trained distance runners may have normal sinus rates of 35 to 40 a very slow pulse usually signifies heart block, more often atrioventricular than sinoatrial If the rate is shown to be quite steady in the thirties and the patient feels well no specific therapy is needed but if there are faint attacks, with or without syncope and convulsions more study and treatment are indicated. In severe cases of the Adams-Stokes syndrome (slow pulse with attacks of syncope and convulsions) it is very important to obtain an electrocardiogram during an attack If the finding is simply that of ventricular or total heart standstill epinephrine is the therapy of choice given as often as may be needed. But if *ventricular fibrillation* is the answer then epinephrine is to be avoided since it can itself sometimes induce such an arrhythmia

Now let us consider electrocardiographic clues that may

be found without relationship to arrhythmia that is which occur with either normal or abnormal rhythm First as to the effect of *heart position* Before the days of the routine six precordial leads it was often difficult to tell from the original three limb leads whether *right axis deviation* of slight degree giving an angle say of +95 degrees, or a bit more meant a *vertical heart position* which it frequently did or *enlargement of the right ventricle* Of course a high degree of right axis deviation or the presence of very prominent *P* waves accompanying right axis deviation was taken correctly as evidence of right ventricular enlargement due either to congenital pulmonary stenosis or atrial septal defect, mitral stenosis, or the cor pulmonale A high degree of right axis deviation with normal rhythm favors the diagnosis of one of the congenital defects over that of mitral stenosis which with much right axis deviation practically always shows atrial fibrillation also In congenital heart disease the presence of right bundle branch block favors the diagnosis of an atrial septal defect. Nowadays we have in the chest leads very helpful clues as to ventricular size and details of atrial action In the case of right ventricular enlargement of more than the slightest degree the *R* waves over the right ventricle that is, in Leads V V_R and V and also in the V leads 2 and 3 to the right of the sternum have more amplitude than normally when they are small Figure 19 shows two records of right axis deviation in the limb leads, the first due to a vertical heart position and the second to right ventricular enlargement. Of course other factors too may influence the height of the complexes, especially the distance of the electrode from the heart therefore an obese chest wall or pulmonary emphysema or pleural or pericardial effusions reduce the amplitude of the electrocardiographic complexes, while a lean chest wall enhances it.

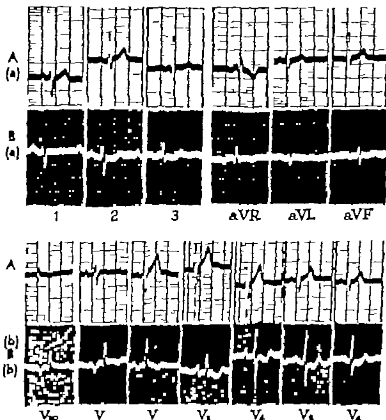


Fig. 19 (A) Electrocardiogram (13 leads 1 2, 3 VR, VL, aVF V ... d V) sho ing right axis deviation in the case of normal ertical heart. J P male ged 17 5 feet, 11 inches, 165 pounds.

(B) Electrocardiogram (13 leads. 1 , 3 aVR, aVL, aVF V V V V V and V₆) showing right axis deviation with right entricular hypertrophy in case of mitral stenosis, showi g lso the effect of digitalis. E.P female, aged 29, 5 feet 5 inches, 129 pounds.

A(a)—limb leads A(b)—chest leads, including V_{3R}

B( )—limb leads B(b)—chest leads, including V_{3R}

Left axis deviation in the three classical limb leads requires much the same discussion as does right axis deviation In former days before the precordial leads were introduced we couldn t tell in many cases of left axis deviation of slight to moderate degree that is up to − 30 degrees whether we were dealing with a horizontal heart position or with left ventricular enlargement or indeed with both since they are frequently combined There were however two helpful clues One of these was, and is, *total inversion of Lead 3* that is, with an inverted *P* wave an inverted *QRS* wave and an inverted *T* wave (with correspondingly upright *T* wave in Lead 1)—this means a horizontal heart position The second clue was *inversion of the T waves in Lead 1* accompanying left axis deviation which usually means *left ventricular enlargement* due to systemic hypertension or aortic valve disease though it may also result from coronary insufficiency myocardial disease of other type or pericarditis. The precordial leads help to distinguish between left axis deviation due to a horizontal heart position and left ventricular enlargement inasmuch as in the latter the *R* waves are of higher amplitude than normal over the left ventricle, that is, in Leads V V₅, and V while th *T* waves tend to be low flat, or even inverted. The same qualifying statement applies here that was made in the case of the precordial leads over the right ventricle the nearer the heart to the electrode the larger the complexes since chest wall fat, pulmonary emphysema and pleural r pericardial fluid decrease their amplitude Figure 90 shows two records of left axi deviation the first due to a horizontal heart position without heart disease and the second due to left ventricular enlargement.

When both ventricles ar enlarged their effects on the limb leads of the electrocardiogram may result in a relatively normal measurement of the electrical axis that is

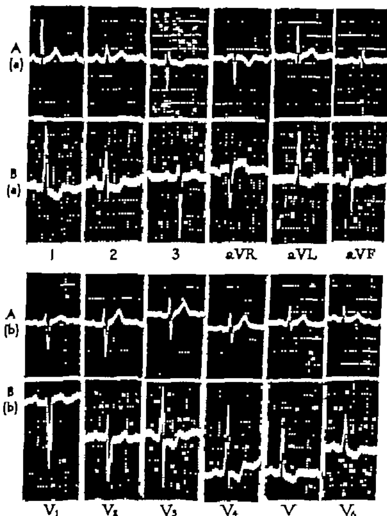


FIG. 20 (A) Electrocardiogram showi g left xis deviation i the case *of a normal horizontal heart.* P.O male aged 34 5 feet 8 inches, 214 pounds.

(B) Left axis deviation i a case of left ventricular hypertrophy (*hypertensive* coronary heart disease, B.P. 210/120 under digitalis therapy). J.M female, aged 44, 5 feet, 160 pounds.
A( ) nd B( )—limb leads A(b) nd B(b)—chest leads.

they may neutralize each other but happily here the precordial leads come to the rescue and usually clearly show the enlargement of both ventricles.

In occasional cases an unusual position of the heart due to rotation around its axes will cause flattening or even

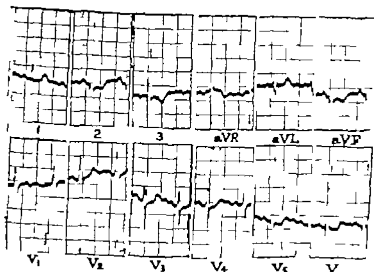


Fig. 21 Electrocardiogram of normal vertical heart with inverted T waves in Leads 2 and 3. S.L., female, aged 17, 5 feet, 3 inches, 120 pounds.

inversion of the *T* waves, most commonly in Lead 2 but also even in Lead 1 and in the precordial leads, resembling at first glance the effects of coronary atherosclerosis or cardiac enlargement. For example, a vertical heart position with low diaphragm may be attended by inversion of the *T* waves in Leads 2 and 3 largely corrected (especially in Lead 2) by elevation of the diaphragm by deep expiration (Fig. 21). It should be added that the *T* wave of course is normally inverted in Lead aVR and not infrequently also

in aVL or aVF The clue to the effect of position is to re peat the electrocardiogram in different positions of the body and of the diaphragm in doubtful cases

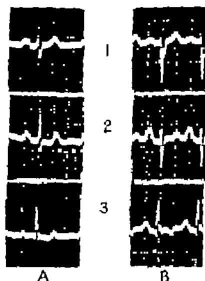


Fig. 22. (A) Elect ocardiogram showing wide P wa es due to l ft t ial enlargement i case f mitral stenosis. M R., female aged 30 (B) Electrocardiogram showing high P wa es associated with right trial nd entricular enlargement n congenital heart disease F W male, aged 26

It is also evident that the degree of right axis deviation is much greater is the rul in the case of congenital heart disease tha in that of mitral stenosis. Limb leads 1 2, and 3

Atrial enlargement is often indicated by increased size of the *P* waves. Leads 1 and ⁹ and higher precordial leads than those routinely used and placed at the level of the third intercostal space over and close to the sternum show the abnormal *P* waves best. In Lead 1 for example there are not infrequently broad not necessarily high *P* waves due to the *left atrial enlargement* of mitral stenosis while

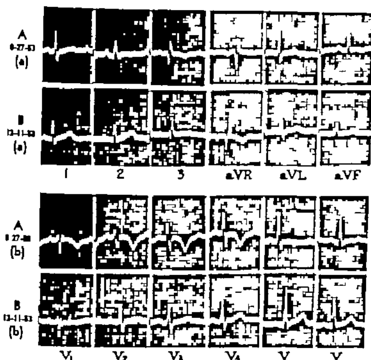


FIG. 23 Electrocardiogram of case f acut or pulmonale due t massive pulmon ry embolism R.P male aged 41

(A) during maximum effect A( )—limb leads A(b)—chest leads
(B) fter recovery B( )—limb lead B(b)—chest leads

the *P* waves of *right atrial enlargen ent* due to certa n congenital defects tend to be high rat er than broad (Fig 22).

An mportant clue to the acute enlargement and anoxia of the right ventricle due to massive pulmonary embolism and alled the *acute cor pulmonale* is to be found in the electrocardiogram The electrocardiogram may show thi pattern only transiently often for but an hour or two rar ly for days, and so it is important to secure a record

as near the time of the height of the trouble as possible and to repeat it several times if necessary for comparison of serial records. The typical pattern consists of the new development or of the increase in amplitude and duration of an *S* wave in *Lead 1* and of a *Q* wave in *Lead 3* of the

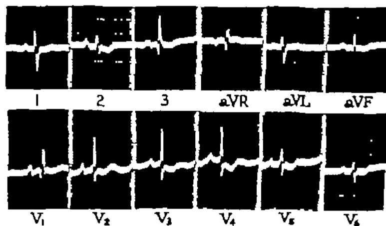


Fig. 24 Electrocardiogram of case of chronic cor pulmonale No digitalis effect. D R., male, aged 40 5 feet 5 inches, 155 pounds

flattening or inversion of the *T* waves in Lead 2, of the inversion or increased inversion of the *T* waves in Lead 3 and of inversion of the *T* waves in the precordial leads over the right ventricle V and V (Fig 93). Chronic enlargement of the right ventricle in a case of *chronic cor pulmonale* of high degree is clearly revealed in the electrocardiogram (Fig 24).

Now we come to the most important reason of all to take an electrocardiogram, namely to determine the presence or absence of clues denoting *coronary insufficiency* or scarring of the heart by *myocardial infarction*. The use of the electrocardiogram for this purpose transcends its use for the study of all other conditions added together for three

reasons 1) coronary heart disease in civilized communities has become the commonest kind 2) it is always of great immediate importance because of its hazard and 3) it may be discovered only by electrocardiogram or at least confirmed thereby in doubtful cases with obscure history It should however be recognized that coronary heart disease may be present with no evidence of it in the electrocardiogram, even after exercise like the so-called two step test of Master or during the hypoxia test of Levy as in a case with old coronary occlusion little or no scarring therefrom and no current coronary insufficiency

Coronary insufficiency of more than very slight degree transiently as in the case of angina pectoris caused by effort or excitement or prolonged even for hours as in the case of acute coronary thrombosis whether or not myocardial infarction ensues almost invariably shows itself in the electrocardiogram It tends to cause a considerable displacement of the S-*T* segments or in the chest leads overlying the region of anterior myocardial anoxia a considerable S-*T* segment elevation as in the case of acute anterior myocardial infarction illustrated by Figure 25 Acute posterior myocardial anoxia on the other hand may cause a transient depression of the precordial S-*T* segments the usual acute posterior myocardial infarct shows relatively normal chest leads and elevated S-*T* segments in leads 3 and aVF as in Figure 26. A slight depression of the S *T* segments and a lowering of the *T* waves is, however a normal physiological reaction associated with the tachycardia caused by exercise Unhappily on occasion this physiological reaction has been wrongly interpreted in a healthy man as evidence of coronary heart disease and a severe cardiac neurosis has resulted I have encountered a number of such cases.

Also of course the depression of the S-*T* segments and *the lowering to inverting of the T waves due to the effect*

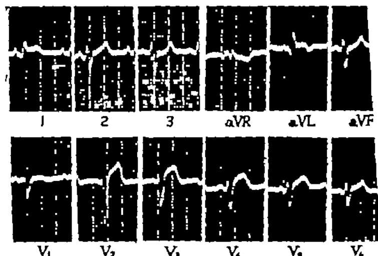


Fig 25 Electroc dlogram of a case th n acute anterior myocardia infarct (Infarct 8/8 53, ecg taken 8/10/53.) N C., male, aged 44

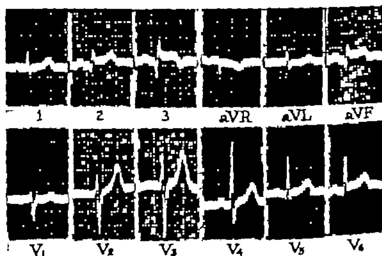


Fig. 26. Electrocardiogram of case with an acute posterior myocardial I farction (Infarct 11/6/51 ecg taken 11/7/51) H B male aged 48.

of digitalis (Figs. 21 and 27) lasting f r da s eeks r yea while the drug is being taken must be carefully distin guished from the effect of coronary insufficiency hich differentiation is usually easily d ne years ag Ho e er there was a noted trial in New York City of a gr up of c

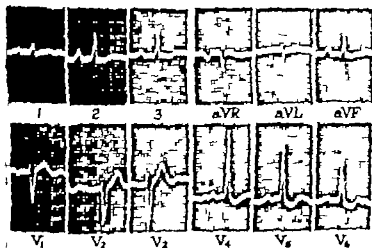


FIG. 27 Electroc diogr m showing sagging of S T segment due t digitalis eff t also norexi and di urbed sion (Had been taking digitoxi 0 mg q 2 day bec use previous dosage q d used flickering, purplish sion, nd norexia. Dosage educed fter this tracing t 0 1 mg 6 times eekly Coronary heart disease) J 1 male aged 59

spirators who had illegally secured large sums of disability insurance by using digitalis to mimic coronary heart disease electrocardiographically

Th e c r nary insufficiency attended by angina pectoris and th e longer lasting myocardial anoxia caused by acute coronary thrombosis without myocardial infarcti n usually subs de ithout leaving any new residual abnormality of the lectrocardiogram but if a *myocard al scar* of any ap-

preciable size results there are changes left in the electrical record which are very helpful clues to past happenings of the sort. For example if the *scar involves the anterior wall of the left ventricle* close to or including the septum which is a very common location Q waves are likely to appear in

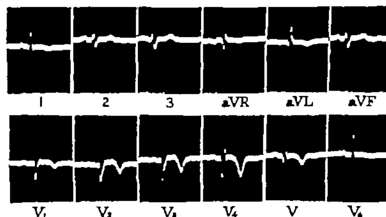


FIG. 28 Electrocardiogram showing evidence of large nteroseptal myocardial i farct which had occurred three mo ths earlier A.G. male, aged 62.

Leads 1 and aVL and the *R* waves disappear in the chest leads V V and often V which overlie the infarcted area (Fig 98) If the scar is very extensive Q waves are found also in Leads V and V or the *R* waves may disappear in those leads, the infarct extending far laterally A lateral infarct may show changes only in the most left of the pre cordial leads V and V or V and V (and in limb leads I and aVL) Sometimes there remain only a few tell-tale Q waves over the site of the well healed scar (Fig 99) The chest leads have become very important for locating, in fact almost pinpointing, the damaged heart muscle all six or *indeed even seven or eight precordial leads* should be taken the old Lead 4 and the two or three routine pre-

cordial leads taken later in electrocardiographic history are quite inadequate in many cases.

If on the other hand the scar involves the *posterior wall of the left ventricle* which is also a common (though slightly less common) site a Q wave usually appears, or

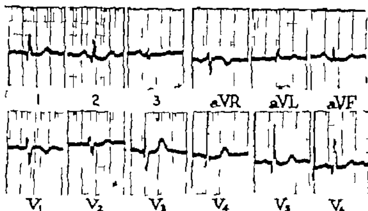


Fig. 20 Electrocardiogram showing evidence of well healed anterolateral myocardial infarct, which had occurred [illegible] years earlier (note prominent Q waves in Leads 1, 2, aVL, V [illegible] V [illegible] and V [illegible] with normal T waves) E.J.F. male aged 51

grows larger if already present in Leads 2, 3 and aVF (Figs. 30 and 31) and if the scar is large enough to include the lateral wall a Q also appears in Leads V and V. If it were a comfortable thing to do to take an esophageal record one would find therein absence of the R waves over the scar. The precordial leads are usually normal in a case with a posterior left ventricular infarct of average size except during the initial process when the S-T segments may be much depressed (in contrast to their elevation in a case with an acute anterior infarct).

Some infarcts are so large that they involve both anterior and posterior walls of the left ventricle or there may be

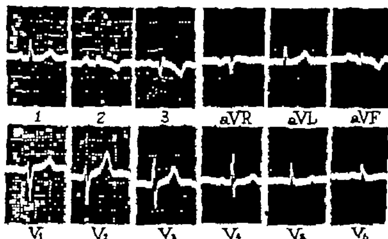


Fig. 30 Electrocardiogram showing evidence of subacute posterior myocardial infarct (infarct March, 1952, ecg taken June 1952). V.L. male, aged 54

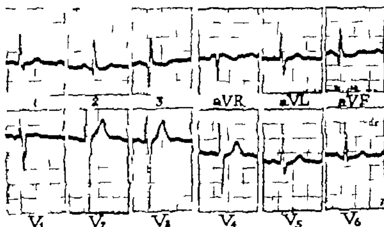


Fig. 31 Electrocardiogram showing evidence of well healed posterior myocardial infarct which had occurred 2 years earlier (note prominent Q waves in Lead 2, 3 and VF). L.W. male aged 47

separate infarcts in front and in back (often at different dates) In such cases the electrocardiogram may be equivocal and not clearly diagnostic since there is a balancing of the effects with a neutralization of the typical changes caused by both lesions. In such cases the clinical picture is more diagnostic than the electrocardiogram and is to be relied upon On the other hand some infarcts are so small that they may affect only one chest lead and that lead may not be one of those routinely taken but higher on the chest or further out in the axilla.

It is very important to know that in a case of acute coronary thrombosis the electrocardiogram may not show characteristic changes for a good many hours or even several days and therefore it is essential to take at least several serial records. Even after the clinical diagnosis has *been confirmed and the location of the infarct established* serial electrocardiograms taken perhaps once a week for a month or two are often very helpful in the follow up In closing this discussion of the electrocardiogram in coronary heart disease I would add what has been mentioned earlier in reference to disorders of rhythm namely that three disturbances are commonly caused by coronary insufficiency *or occlusion although they are not diagnost n* they can result from other causes t they are ventricular paroxysmal tachycardia a v block and bundle branch block

The myocardium may be affected by other diseases than that caused by coronary atherosclerosis Such *myocarditis* whatever its cause can produce electrocardiographic changes that are usually nondiagnostic There may be very low voltage of all the complexes P QRS and T waves in the limb leads and sometimes even in the chest lead there may be simply abnormal (chiefly inverted) T waves there may be heart block, either atrioventricular or intraventric

ular (bundle branch) block or both (Fig 32) or there may be ventricular paroxysmal tachycardia or atrial flutter or fibrillation. If there is widespread disease of some sort elsewhere in the body such as lymphoma or amyloidosis or anemia an abnormal electrocardiogram may in the absence of evidence of an unrelated specific disease of the

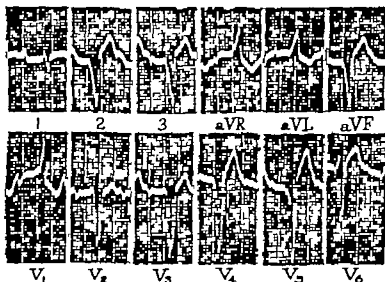


FIG. 32 Electrocardiogram of case of extensive myocardial disease of unknown origin, confirmed by autopsy (note both a-v and bundle branch block) R.G male, aged 21 6 feet, 160 pounds.

heart, mean cardiac involvement by the particular systemic disease. Sometimes there may be a localized lesion such as an echinococcus cyst or a new growth which may give localized evidence of its presence in the electrocardiogram resembling somewhat the effect of an infarct.

Pericarditis both acute and chronic, can affect the electrocardiogram in large part doubtless due to the subjacent myocardial involvement. There may be for a few days an

elevation of S-T segments in the limb leads (Fig. 33) and in the precordial leads too subsiding later to leave no trace, or with a large effusion there may be generalized low voltage. Chronic adhesive pericarditis of slight degree may produce no changes but chronic constrictive pericarditis, which means involvement of serious degree always affects the electrical record with extensive T wave changes

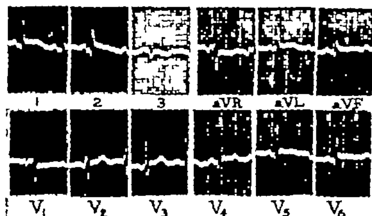


Fig. 35 Electrocardiogram showing the effect of acute pericarditis, diagnosis proved at postmortem examination. Note the elevated S-T segments and flat T waves in Leads 1, 2, aVL, aVF, V[illegible] and V[illegible]. M.S., male, aged 58

but fairly normal QRS waves (Fig. 31). In such cases atrial fibrillation is often present also and in a few there may be evidence of right ventricular enlargement due to preponderant constriction of the left ventricle so giving rise to an electrocardiographic pattern closely simulating that of mitral stenosis with atrial fibrillation

An interesting and sometimes very useful electrocardiographic clue is that in *myxedema*. In an advanced case the T waves are flat with low voltage of the QRS waves also

(Fig 35) This abnormality is different as a rule from the various patterns found in other conditions or diseases, such as coronary heart disease and yet I have encountered several errors in diagnosis based on misinterpretation of the

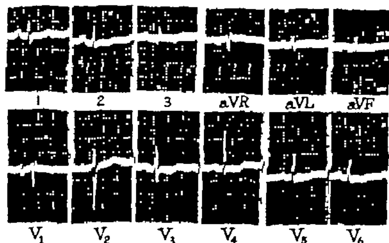


Fig. 34 Electrocardiogram showing the effect of chronic constrictive pericarditis, confirmed by operation Note the very low flat or diphasic T waves in all 12 leads with relatively normal QRS waves W V male aged 31

lectrocardiogram with inadequate general clinical appraisal

Finally various drugs and toxins may affect the electrocardiogram The influence of digitalis has already been mentioned (see Fig 27) Adrenalin in large dosage can invert the *T* waves in Lead 2 Quinidine sulphate can produce bundle branch block and depress the pacemakers. Tobacco can lower the *T* waves, perhaps through its sympathetic nerve action Carbon monoxide can cause a v block. Electrolyte imbalance can cause changes. Low calcium content in the serum prolongs systole High potas-

sium levels (*hyperpotassemia*) cause high pointed *T* waves delayed a v conduction and widening of the *QRS* waves culminating in atrial paralysis, gross bundle branch block, and deformed S-*T* segments and *T* waves. *Hypopotassemia*

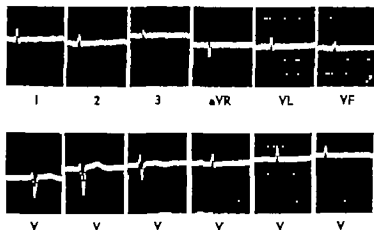


Fig. 35 Electroc rdiogr m show ng flat *P* es nd low ltage of the QRS w es i typic l myxedema A D femal ged 65 B M R = −36%

also distorts the record w th depression of S-*T* segments and *T* wa es prolonged *Q T* duration nd large *U* waves Incidentally negati e *U* waves usually indicate the presence of heart disease

CHAPTER 13

THERAPEUTIC CLUES

THE CHIEF clue of course, to the proper treatment of a patient with disease or disorder of the heart and great vessels is the correct diagnosis but there are details of diagnosis and sometimes multiple conditions of varying degrees of importance that require considerable skill in their therapy In the preceding chapters certain of the many therapeutic clues have been mentioned but for convenience emphasis and more comprehensive consideration they are gathered together in this last chapter and others added.

There are several observations about treatment in general that are worthwhile presenting before taking up the individual clues The first is that it is always wise to get along with as little treatment as possible in reaching the desired goal Certainly one of the errors of the past which is still too often encountered is overtreatment. Even though each thing done can be approved individually in a given case, it may not be necessary to use all or even several of the measures available· one or two may suffice. Wise judgment from this angle can often save a patient the strain of excessive medication the too early use of some therapy which may be needed more later and the additional drain on his pocketbook.

A second point is that now and then a rest day or even several days with no medicine at all may be very helpful especially if the patient is having toxic effects from drugs or is tired out from their effect, either physically or mentally or in need of re-evaluation as to their necessity On rare occasions I have found such therapeutic holidays lifesaving

A third point concerns the polypharmacy that happily has been on the wane during the last generation but which still exists in certain regions or countries. The complicated shotgun prescriptions of former days are now infrequently encountered in medical centers but their place is sometimes taken by a multitude of drugs including vitamins which are given separately so that a patient may have a shelf or a drawer full of bottles from which he takes pills, capsules, powders or drops at frequent intervals all through the day and night. Here should be included an indictment of much of the parenteral (hypodermic intramuscular and intravenous) therapy of various parts of the world which should be free from dependence upon such a method of administration of the medicines.

A fourth point has to do with the trade names of many of the more important drugs. Often various drug companies have their own pet names for the same derivative of a commonly used drug such as digitalis or one of the new popular blood pressure medicines, and this may and sometimes does lead to confusion. It is important for the physician to keep himself posted about these names or else better still to use the standard names for the preparation with, if desired, the name of a pharmaceutical house or its trade name for the drug in parenthesis after it. Thus digitoxin is the same as Digitaline and Purodigin

Finally it is generally helpful to explain to the patient himself what the various medicines and other therapeutic

effects actually are and why they are being employed. It is half the battle to secure the understanding and coöperation of the patient, especially when it is likely that years of treatment lie ahead.

Now let us take up the clues to therapy themselves under various headings.

Myocardial weakness and failure. Probably more knowledge and skill and experience are needed in handling heart muscle weakness and failure than almost any other complaint in the practice of medicine. The very first evidence of myocardial failure demands attention, whether it involves the left ventricle first, as it usually does because of the frequency of systemic hypertension, aortic valve disease, and myocardial infarction, or the right ventricle first as may happen with mitral stenosis or certain congenital defects. The clues to the beginning of left ventricular failure already mentioned are 1) too easily induced dyspnea, 2) pulsus alternans, 3) apical gallop rhythm, and 4) accentuation of the pulmonary second sound, while the right ventricular failure clues are 1) beginning engorgement and pulsation of the jugular veins in the sitting position, 2) tenderness and slight enlargement of the liver, and 3) a diastolic gallop at the lower end of the sternum.

Two measures of therapy are most useful at the very start of the failure. Often they suffice if well applied without the need of resorting to other more radical treatment which is often a bother. The first of these measures is *rest*, more or less complete, for as long as necessary until some myocardial reserve has been regained; two or three weeks may suffice. Complete bed rest is usually not necessary and as a matter of fact is often unwise. To be up and around, sitting in a chair much of the day and with bathroom privileges, is the best program for most patients. Much conversation with family or visitors and more than

essential telephoning are to be avoided After this initial saturation with rest, rations of rest daily weekly monthly and yearly should be arranged.

The second measure of equal importance is the use *of digitalis* which is the most helpful medicine for a weak heart—nothing better has as yet been found. The drug should be given to full but not toxic saturation (digitaliza tion) slowly that is in the course of a few days to a week unless an emergency arises, and its effect maintained as a rule permanently that is, for the rest of the patient s life To digitalize too rapidly that is, in a few hours or even in one day is usually unnecessary and often unwise since it may upset the patient who should not be disturbed by this medicine which he may have to take for the rest of his life and an antagonism to which should be carefully avoided even its taste might then become repugnant. There are many different good preparations of digitalis and many different ways of giving them I have found in the great majority of patients tl e following plan to work well digitalis leaf 0 065 gram or 1 grain pill or tablet t i d for a week and then once a day thereafter Or one of the puri fied preparations may be given in much the same way *e.g* Digitoxin 0 15 mg t i.d. for a week and once daily there after A few patients need a little larger daily dose than this or a little smaller for example a tablet of 0 1 gm or 1½ grains of the leaf or 0.2 mg of digitoxin or 0 0. gm. or ¾ grain of the leaf or 0 1 mg of digitoxin A few patients requ re two three or even four times the average dose every day for months or years to maintain an adequate effect on heart rate or tone or both "There is only one ontraindication for the use of dig talis, namely to use it when it is not needed.

If these two measures do not suffice (and as a matter of fact tl ere are relati ely few patients where they do not,

except near the end of their lives) then we have other resources, in particular *reduction of sodium intake* in the diet and the administration of *mercurial diuretics* and in very rare cases the use of irradiated iodine To be sure excessive salt intake is wisely curtailed early in all cases of cardiac strain and failure but considerable or extreme salt restriction is so unpleasant that it is well not to apply it until really necessary The same statement holds true for the mercurial diuretics whether given parenterally or by mouth they are invaluable when needed and then given weekly or every few days or even daily for awhile they can save and prolong lives but they can also on occasion be not only very uncomfortable but actually dangerous by depleting the patient and favoring the occurrence of the low salt syndrome (prostration and resistance to therapy) which in turn requires the replacement of sodium Mercurial diuretics should be used sparingly when the intake of salt is very low Now and then one can use helpfully a preparation of resin *fortified with potassium to avoid* excessive loss thereof to prevent some of the sodium in a more palatable diet from being absorbed in the gastrointestinal tract.

In edematous patients resistant to diuresis aminophyllin intravenously can be effective in helping to clear away the congestion but it must be used with care to avoid electrolyte depletion. On the other hand it is important to remember that morphine is an antidiuretic agent.

Very infrequently does one need to resort to more radical therapy but a clue to the possible value of such is the chronic persistence of failure despite the application of the measures cited above. Then the administration of *irradiated iodine* may have a place to reduce the basal meta

I hypertensi patients th Intractable congesti failure the addition of "hypotensi e drug may w the day

bolic rate of the body and its demand for oxygen thus easing the strain on the heart. One must figure however on a delay of six weeks to three months for adequate effect of this sort from adequate dosage up to 50 or even 75 millicuries in one or several doses may be needed. The use of *Southey's tubes* to draw fluid from the edematous legs (or even the scrotum) is a desperate but at times helpful measure also. Both of these procedures have been very useful in patients of my own. Another treatment which I have not employed but which has been recommended in desperate cases of intractable congestive heart failure is ligation of the inferior vena cava; surgical operations are however wisely avoided as a rule in such cases.

Finally in this discussion of myocardial failure mention should be made of an important clue to some kind of a serious complication. That clue is the very obstinacy or intractability of the congestive failure itself. Often it means that there is present, perhaps obscurely, a previously unrecognized or new complication such as active infection, an example of which is rheumatic fever, embolism or infarction elsewhere as in the lung or heart, and thyrotoxicosis. One should search for such conditions. It is important to remember that in patients with myocardial failure death usually results from a last straw such as pulmonary embolism and not from the failure itself.

Coronary insufficiency. So soon as it is evident that insufficiency of the coronary circulation is present because of the symptom of angina pectoris whether or not there is any abnormality of the electrocardiogram one should take action. It should not be treated lightly or casually but neither should it be viewed with alarm or pessimism. It is, as a rule, best to take the patient wholly into one's confidence, explaining what the condition is, that it must be treated with respect, and that recovery or at least consider-

able improvement is, if not certain at least likely provided certain measures are carried out.

When some particular exertion or excitement causes the discomfort of angina pectoris that exertion or excitement should be avoided for as long a time as necessary It may take weeks or months or even a year or two for the angina to abate and normal full or at least increased activity to be resumed the inculcation of patience is therefore an essential part of the treatment in the majority of cases In general it is wise to tell the patient and his family that he should avoid hurry unnecessary worry overexertion overeating (at any one time or in total number of calories in any one day) and exposure to severe weather (intense cold, storms and strong winds in particular) The amount of restriction will depend, of course on the degree of trouble If the attacks are very easily induced several times a day the patient had best rest up at home (or in a hospital) for a few weeks away from his business and exciting visitors or telephone conversations. Usually he is better up and around quietly rather than in bed. The nitrites are the only medicines, except perhaps for mild sedatives, that are of general value in such patients Contrary to much of the current custom I do not find or recommend the use of alcoholic drinks as particularly helpful they are far inferior to the nitrites as medicine and although in small to moderate dosage often pleasant and relaxing, they favor overweight and at times even alcoholism Only in chronically severe cases of this sort lasting six months to a year or more is it wise to recommend radical treatment I have found that the best radical therapy in such cases is the reduction of the basal metabolic rate by the administration of irradiated iodine

Angina pectoris at rest that is, *decubitus* is especially important and demands great respect. Actually in many such cases coronary thrombosis has taken place even if

patients particularly is rest at home (or in the hospital) for there has developed no myocardial infarction. In these some weeks highly to be recommended but not in bed unless the patient feels better there or infarction develops. Nitrites may be freely used. Anticoagulants to try to prevent further thrombosis are being tried in some cases but we haven't yet the final answer as to their value in this respect

In days gone by angina pectoris and coronary thrombosis *were viewed with such dread and pessimism that many* patients, feeling fatalistically that their days were numbered anyway ignored precautions and did often come to grief Now we have learned that recovery even complete is not at all uncommon even in patients with the most severe angina pectoris decubitus, provided they are careful to avoid strains during the weeks or months of their disability This advance in our knowledge and the resulting change in the attitude of many patients have constituted one of the most encouraging developments in our treatment of coronary heart disease during the last generation

In patients with actual *acute myocardial infarction* (secondary to the coronary thrombosis) as indicated by the clues of fever leukocytosis, and characteristic electrocardiographic changes, much the same treatment as mentioned above is to be recommended plus more complete rest for the first three weeks, preferably in bed the use of anticoagulants for several weeks in the majority of sicker patients under hospital care and therapy of any complications that may arise, such as myocardial failure Again it is very important at the start of the treatment to explain to the patient just what is going on so that he will gladly cooperate in the program and not rebel at the sentence imposed. Prostration with actual shock during the first few hours of acute coronary thrombosis is a bad prognostic

sign but a good many cases recover from this either with routine treatment or with the added help of neosynephrin or mephentermine sulphate transfusion of whole blood or packed red cells has also been thought to help but its value is still open to question. The duration of the total convalescence from several weeks to several months should naturally depend on the amount of the heart muscle damaged and on the presence or absence of complications. A complete recovery with little or no cardiac enlargement and only residual evidence of the scar in the electrocardiogram is a clue in most cases for the resumption of a normal active life

Disorders of cardiac rhythm Since by far the commonest disorder of rhythm is the *premature beat or extrasystole* since this is found usually in healthy people without heart disease and since it causes no harm and in many persons is not even noticed by them no treatment at all is necessary in the great majority of individuals who show this arrhythmia of the heart There are persons however who are bothered by their premature beats whether they have heart disease or not they may feel uncomfortable either due to the early contraction itself which may be felt as a throb in their neck or the pause when their heart seems to stand still or the forceful thump of the afterbeat Persons troubled by such sensations may have uncomfortable days or nights and so deserve some treatment. The simplest measure of all which alone is often effective, is full reassurance and a statement that most persons become so accustomed to the arrhythmia that eventually they don t feel it at all Sometimes it is obvious that there is a particular exciting cause of the extrasystoles, such as tobacco coffee tea alcohol indigestion or unusual excitement or fatigue control of that particular factor may then prove to be sufficient to abolish or reduce the palpitation Infrequently drugs are

needed but they should never include narcotics. Quinidine sulphate in tablet form 0.2 gm or 3 grains each, may or may not be effective when taken several times a day often it is not necessary to take it every day but only on occasion when the arrhythmia is more disturbing Now and then a sedative, particularly such as phenobarbital in small dosage may work well or even bromide which was recommended years ago by Sir James MacKenzie who found however that the best treatment consisted of reassurance and exercise in the open air

Paroxysmal tachycardia may be as unimportant as extrasystoles and may be treated in exactly the same way as outlined above On the other hand it can be much more disagreeable and in the presence of heart disease even harmful and in rare cases dangerous therefore in such cases more than simple reassurance should be applied. At the beginning of an attack of atrial tachycardia pressure on the carotid sinus may be tried it is effective in perhaps t 10 per cent of cases. If it is ineffective I have found that the administration of two tablets of quinidine sulphate repeated in one or two hours if the attack is prolonged has shortened the paroxysm in many of my cases. Syrup of ipecac may work too but it is itself disagreeable Mecholyl and pronestyl may be tried in more obstinate cases and in a few patients digitalization and its maintenance have been found very useful If the paroxysmal tachycardia is of ventricular type quinidine and pronestyl are the drugs of choice. The most important clue to the best treatment is the identification of the mechanism at fault by the electrocardiogram which can be helpfully repeated in the follow-up of the therapy of any arrhythmia.

Atrial flutter and fibrillation also best identified by electrocardiogram, are more advanced disorders of rhythm and more often found in cardiac patient than in normal

persons. Although reassurance is still in order in most cases *it often needs to be qualified according to the extent* of the underlying heart disease. For paroxysmal or newly developed atrial fibrillation the restoration of normal rhythm by quinidine is often to be recommended and is frequently successful if however the paroxysms often recur or atrial fibrillation has been present for years the treatment of choice is digitalization and its maintenance For atrial flutter I have found digitalis preferable to quinidine from the very start the ventricular rate can be reduced thereby through increasing the grade of block from 2 to 1 to 4 to 1 or thereabouts or atrial fibrillation may evolve or normal rhythm return It has also been stated that full doses of atropine after digitalization may abolish atrial flutter

As in the case of myocardial failure so in the case of atrial fibrillation or flutter a refractory resistance to treatment with maintenance of too fast a heart rate despite ample digitalis may be a clue to the presence of some important complication such as infection (e g rheumatic fever) infarction (pulmonary or myocardial) or thyrotoxicosis. *It is important, however to recognize that with* atrial fibrillation, much increase of heart rate on exercise means that the patient is inadequately digitalized even though the heart rate at rest seems to be well controlled in the case of the complications just mentioned the heart rate tends to be rapid even at rest.

Next, we come to *heart block Bundle branch block* is a clinically silent condition and needs no treatment Even most cases of *a v block* and *s-a block* need no treatment themselves unless symptoms of dizziness or faintness or *actual syncope with or without convulsions (Adams-Stokes* syndrome) develop when the heart rate drops to 20 or less per minute or standstill occurs for 8 to 10 seconds.

Then the drug of choice is epinephrine hydrochloride 1 1000 solution ¼ to 1 cc. parenterally as often as needed if ventricular fibrillation has been ruled out by electrocardiogram as a cause of the lack of ventricular pulsation. In milder cases ephedrine may have a favorable effect.

In conclusion of this chapter a few observations may be added concerning a few other emergencies due to disease of the heart and great vessels. *Acute pulmonary edema* is commonly due to heart disease although there are other causes. Important clues as to its treatment and also to the prevention of future attacks depend on the identification of the kind of heart disease that underlies it and on the discovery of the factor or factors that have precipitated it. However no matter what the cause there is one more or less specific immediate therapy for the great distress that practically always accompanies the pulmonary edema, and that is the parenteral administration of a narcotic mor phine sulphate is the narcotic customarily used and generally it is very helpful It is also wise usually to administer oxygen if it is available But that is not enough in many cases. If one is dealing with *sudden failure of the l ft ventricle* due to hypertension aortic valve disease or myocardial infarction it is important to administer digitalis in full dosage within a relatively few hours if the drug is not already being taken in adequate amounts. In such cases digitalis in purified form in solution may wisely be g en intravenously half the digitalizing dose at once to be followed at four hour intervals by the third and fourth quarters of the total dosage respecti ely varying the program somewhat as circumstances demand. Thus dig xin may be injected in doses of 0 8 mg 0 4 mg and 0 4 mg. respecti ely or digitoxin in doses of 0 6 mg. 0 3 mg and 0 3 mg. respectively Once acute pulmonary edema has been set off in such cases *full digitalization* should be main-

tained ever after If the edema does not subside in a few hours mercurial diuresis may be added helpfully to the treatment And of course rest with head elevated and limitation of sodium intake are also to be prescribed Rarely is it necessary to resort to venesection or compression of the limbs by blood pressure cuffs in the treatment of acute pulmonary edema.

If however the basis factor is *mitral stenosis* with a superimposed tachycardia there should be instituted along with the opiate injection treatment as specific as possible for the fast heart rate If the tachycardia is of sinus type its cause whether infectious toxic, or emotional should be combatted by antibiotic antitoxin, or sedation respectively If the tachycardia is paroxysmal atrial or ventricular or due to fibrillation or flutter the treatment should be directed thereto with quinidine or digitalis or other therapy as already described earlier in the chapter Prevention of such tachycardia in the future by appropriate measures should be planned at once on the subsidence of the present attack and in many cases of mitral stenosis who prove to be suitable subjects mitral commissurotomy may be a valuable protection for years to come

A cardiovascular emergency that frequently complicates heart disease and that in itself may precipitate acute pulmonary edema is *pulmonary embolism* After the immediate treatment of the attack of edema as described above has been given, an effort should be made to prevent further episodes of the sort Actual pulmonary embolectomy once suggested and tried has proved to be neither feasible nor wise but it may be possible to ward off further trouble by two procedures 1) ligation of the leg veins if there is any suspicion of thrombosis therein and embolism from right

heart chambers seems unlikely and 2) the use of anticoagulants.

Constrictive pericarditis demands when *acute* due to tamponade, evacuation of effused fluid and when *chronic* pericardial resection These measures are often lifesaving and may reestablish complete health and full longevity

Subacute or acute bacterial endocarditis or even strong suspicion thereof demands antibiotic therapy which is today usually curative It is best to start with and to continue penicillin in large dosage but *in vitro* testing of the effect of the various antibiotics on the causative organism should be carried out at the very start for future guidance of the therapy which requires a minimum of at least a month

Finally among the more important emergencies due to diseases of the heart and great vessels dissection of the aortic wall (*dissecting aortic aneurysm*) should be considered Though uncommon it is very serious Morphine and the treatment of a state of shock if present are all we have to offer but the correct diagnosis should be made because such is a vital clue to the bad prognosis about 90 per cent of the cases die of rupture within a few hours or days.

APPENDIX

Since the completion of this book I have noted a few important omissions and have received several more clues from various sources which are worth adding in an appendix.

Hemoptysis should have been mentioned specifically as a fairly important clue in Chapter 7 entitled Other Symptoms on page 60 following the discussion of cough and hoarseness. Brisk hemorrhages from the lungs have sometimes been called pulmonary apoplexy and are occasionally seen in the case of patients with tight mitral stenosis but also in cases with bleeding into the pulmonary vessels due to other causes for example, rupture of aneurysms, erosion of vessels by tumors or tuberculosis or other infections. Some patients with mitral stenosis and fragile vessels may bleed from the lungs after excessive effort or from extreme heart rates in paroxysmal tachycardia or fibrillation. Most patients with mitral stenosis who bleed, however have simply blood streaks in the sputum or blood stained froth raised at the time of acute pulmonary edema. This same situation holds in a few patients who develop this phenomenon secondary to acute infarction of the lungs or pulmonary edema resulting from acute failure of the left ventricle due to the strain of acute myocardial infarction or hypertension or aortic valve disease. As a clue therefore hemoptysis, although important in itself must be carefully analyzed since it may be the result either of intracardiac disease disease of the great vessels diseases of the lungs, or intrathoracic disease of other sort.

Similarly from an oversight *examination of the lungs* was not adequately presented in Chapter 8 entitled Signs.

The discussion of the thorax with unusual deformities thereof and pulsations should have included a statement about auscultation percussion and roentgenography of the lungs and pleura

Rales are more commonly due to pulmonary disease than to heart disease Very often they are of no importance at all and represent no disease This is particularly true of the so-called atelectatic rales at the lung bases of many patients who have shallow respiration Deep inspiration opens up the bases of the lungs and may induce for a few breaths crackling rales or even some that seem to be moist Several deep breaths clear these rales however they are not evidence of pulmonary disease or congestion of the lung bases due to heart failure or mitral involvement On the other hand persistent moist rales at the lung bases in patients who have cardiac enlargement and dyspnea and no evidence of pulmonary disease per se may indicate the presence of congestion associated with pulmonary hypertension and failure However such rales are a late manifestation of left ventricular failure dyspnea being far more important as an early indication There are also the localized rales over pulmonary infarcts to be considered and those due to infections of one kind or another Dulness to percussion bronchial breathing and increased density of the lungs on x ray examination are found over areas of the lung which are consolidated as from infarction or infection or compressed by very large hearts pericardial or pleural effusions and tumors.

Hydrothorax is of much importance but must be carefully analyzed If in the case of a cardiac patient the hydrothorax is wholly right-sided or preponderantly right-sided right or total heart failure is to be suspected. If the effusion is limited to the left side some other cause than cardiac is to be looked for: this may consist of acute pleuritis or

pulmonary infarction Thickening of the pleura may cause slight dulness but not the marked dulness of effusion

Friction rubs may be either pleural or pericardial As a matter of fact, not infrequently they are both and are then called pleuropericardial In such cases acute pericarditis is to be diagnosed Once in a while when there is great pressure over the pulmonary artery due to bulging thereof a transient friction rub may be heard even in the absence of actual pericarditis. This has been noted in cases of thyrotoxicosis and of the acute cor pulmonale

Apical heart murmurs that are well heard in listening over the right anterior chest wall suggest an aneurysmal left atrium just as bulging and pulsation of the anterior chest wall to the left of the sternum indicate the probability of hypertrophy of the right ventricle

Resistance and sclerosis in the pulmonary circulation complicating chronic mitral stenosis adds an element of strain such as we find in cases of the chronic cor pulmonale Therefore long standing cases of severe mitral stenosis have something of the chronic cor pulmonale as well as of the direct effect of an increased resistance to the flow of blood through the left side of the heart

Persistence of a rapid sedimentation rate after a good many weeks following myocardial infarction suggests the presence of mural thrombi

The absence of reciprocal changes in Leads I and 3 in the electrocardiogram favors the diagnosis of acute pericarditis over that of acute myocardial infarction.

Heat as well as exercise may be used to test the significance of cyanosis. In cases of right to left shunt both heat and exercise increase the cyanosis.

The low salt syndrome producing weakness and occasionally even confusion is sometimes wrongly called uremia

Dilatation of the aorta and of the pulmonary artery as seen by x ray examination is commonly found in cases of aortic and of pulmonary stenosis respectively

The seriousness of ventricular premature beats in which the R waves fall on the preceding T waves has been pointed out as suggesting the imminence of ventricular tachycardia or fibrillation

On occasion weakness following mercurial diuresis can be prevented by the administration of potassium salts by mouth (3 to 5 grams) following the day of and the day after the injection of the mercury

Finally the use of norepinephrin (arterenol) has been emphasized by a number of my correspondents as being distinctly helpful in some cases of shock accompanying massive acute myocardial infarction.

INDEX

This Book

CLUES IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE

By

PAUL D WHITE M D

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